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ELECTROCARDIOGRAPHY OF THE LEFT VENTRICLE
IN CORONARY ARTERY DISEASE AND HYPERTROPHY

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Thesis submitted for the degree of Ph.D.

To:

Faculty of Medicine
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The Research described in
this thesis was carried out
at the University Department
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Date:

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DECLARATION

I hereby certify that this thesis had been designed,
composed and written entirely by myself and has not been
submitted previously for any degree

Signed: '

Date:

DEDICATION

To my

Mother	Najiyah
Brother	Sherzad
Sisters	Jiyan and Joan

with love and regards

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SUMMARY

This thesis describes a series of studies which were undertaken to improve the diagnostic accuracy of the electrocardiogram (ECG), given the availability of computer assisted measurement techniques. Scalar 12 lead electrocardiograms have been recorded from over 200 cardiac patients recruited specifically for this study. Over 1,500 additional ECGs were available from normal and abnormal test populations. The ECG measurements were correlated with clinical, echocardiographic, radionuclide, coronary angiographic and contrast angiographic data of the left ventricle in order to meet the aims of the study.

The history of electrocardiography was reviewed both with respect to the technique itself and the evolution of equipment through to present day computer assisted technology for recording and measurement of ECG waveforms. In addition, the development of echocardiography, nuclear cardiology and cardiac catheterization was also reviewed with particular attention being given to recent developments in technology which have allowed a reappraisal of gold standards against which the ECG can be compared.

With more specific relevance to the aim of this study, the development of ECG criteria mainly with respect to post mortem examinations was reviewed so that a contrast could be drawn with present day techniques.

In particular, emphasis was laid on the twin areas of left ventricular hypertrophy and ischemic heart disease.

Evaluation of left ventricular function from the scalar electrocardiogram was undertaken in two ways. First of all the latest ECG scoring system of Selvester was assessed, from which left ventricular ejection fraction could be calculated. Secondly, indirect evidence of left atrial overload from the electrocardiogram was also studied. It was shown that left ventricular function could be predicted qualitatively with reasonable accuracy in the early post infarct period (third day) using the 54 criteria/32 points scoring system. However, it was also concluded that this system is of limited value in predicting accurately the ejection fraction from the ECG in individual patients. On the other hand, severe global LV dysfunction (ejection fraction less than 25%) as estimated by Technetium 99 ejection fractions in the early post infarct period (third day) was also predicted by electrocardiographic evidence of left atrial overload as evidenced by an increased P terminal force in lead VI $> 8 \text{ mV.msec}$. This latter finding has not previously been described.

An assessment of the value of the QRS-T angle in the frontal plane as a criterion for ischemic heart disease was also undertaken in a group of patients with isolated T wave abnormalities but without Q wave evidence of myocardial infarction. It was concluded that $\text{QRS-T} > 0^\circ$

alone was of no value as an index of abnormality although in combination with a T wave angle < 0 was highly specific for coronary artery disease. Specificity was assessed on a group of 1,315 apparently healthy individuals.

Echocardiographically determined left ventricular masses indexed to body surface area were utilised as the gold standard for re-evaluation of ECG/LVH criteria. In order to do this, two important steps were taken. First, the effect of measuring echocardiographic parameters using different conventions was evaluated. Substantial differences were uncovered and the conclusion reached that the so called Penn convention should be adopted as opposed to the recommendations of the American Society of Echocardiographers. Secondly, an evaluation of an existing scheme for classifying echocardiograms based on the internationally acknowledged Framingham study was undertaken. This showed that the available methods were unsatisfactory when tested in a sample of over 200 consecutive cardiac patients. A new echocardiographic classification was therefore proposed on the basis of mass and volume. In particular four categories were described, namely - normal, eccentric LVH (increased volume and increased mass), concentric LVH (increased mass and normal volume) and finally a new subgroup termed decompensated left ventricle (increased volume, normal mass).

The remainder of the study concentrated on the

development of ECG criteria for accurately diagnosing left ventricular hypertrophy. Initially a study was made of atypical T wave changes in the anterolateral leads which were found in almost one third of a population with LVH and normal coronary arteries. It was thought that exclusion of such abnormalities from any point scoring system for the ECG diagnosis of LVH would contribute to a reduction of the sensitivity. Therefore a new criterion was introduced in which such changes did contribute to the score if they were associated with high voltages in the same leads. It was subsequently shown that this new criterion contributed to an improvement on the sensitivity of a revised Glasgow point scoring system.

Existing criteria for LVH were reviewed in depth with respect to the new echocardiographic classification system but it was concluded that there was no reliable method by which the ECG could differentiate between concentric and eccentric LVH. In general, in concentric LVH, there was potentiation of the limb lead voltages much more commonly than in eccentric LVH which in turn appeared to augment precordial voltages much more so than did concentric LVH.

The effect of anteroseptal myocardial infarction on the diagnosis of LVH was also assessed. It was found that QS deflections in the anteroseptal leads produced an augmentation of precordial lead voltages when compared with age and sex dependent upper limits of normal.

However there was no effect on limb lead voltages. This finding has not previously been described and led to further modification of the scoring system for the diagnosis of left ventricular hypertrophy and contributed to the improvement with respect to specificity of ECG criteria for LVH in the presence of anteroseptal myocardial infarction.

The crux of the study was the development of a revised so called Glasgow point scoring system for the ECG diagnosis of LVH. This was achieved by modifying the old Glasgow system which was assessed objectively for the first time. The modifications of the old system were based on the results of the correlative studies between ECG, echocardiographic and coronary angiographic data. Application of the new Glasgow point scoring system resulted in a significant improvement in the electrocardiographic diagnosis of LVH with regard both to sensitivity and specificity. This was confirmed in a test population of 500 cases documented by non-ECG methods recorded as part of the European collaborative studies on common standards in electrocardiography. The sensitivity and specificity of the criteria were 65% and 98% respectively. The latter data were thus derived from a truly independent evaluation of the new Glasgow scoring system.

The overall conclusion can be drawn that the availability of computer assisted methods for measurement of electrocardiograms together with recent advances in

technology which allow ECG correlation with cardiac abnormalities can lead to an improvement in the diagnostic value of the electrocardiogram in the fields of coronary artery disease and left ventricular hypertrophy.

1. HISTORICAL BACKGROUND

1.1 EVOLUTION OF ELECTROCARDIOGRAPHY

1.1.1 Electrocardiography in the nineteenth century

The history of electrocardiography and vectorcardiography are well established (Burch and de Pasquale 1964). In 1856, Kolliker and Muller demonstrated the presence of action currents associated with a heart beating. They placed a frog's nerve-muscle preparation in contact with a beating heart and found that the former twitched with each contraction of the heart (Lipman and Massie 1969a).

Marey, in 1876, used the capillary electrometer, which was invented by Lippmann, and photographically recorded the electrical activity of the frog's heart (Macfarlane 1989). Engelman (1878) as well as Burdon-Sanderson and Page (1878) were also among the earliest to plot the potential variations due to the electrical activity of the heart.

Waller (1887), an Aberdeen graduate, published the first known recording of the human electrocardiogram (ECG) as shown in figure 1.1. He carried out his work while he was a lecturer in physiology at St. Mary's, Paddington. He used a capillary electrometer to demonstrate a measurable amount of current in the human body associated with contraction of the heart (Snellen, 1984).

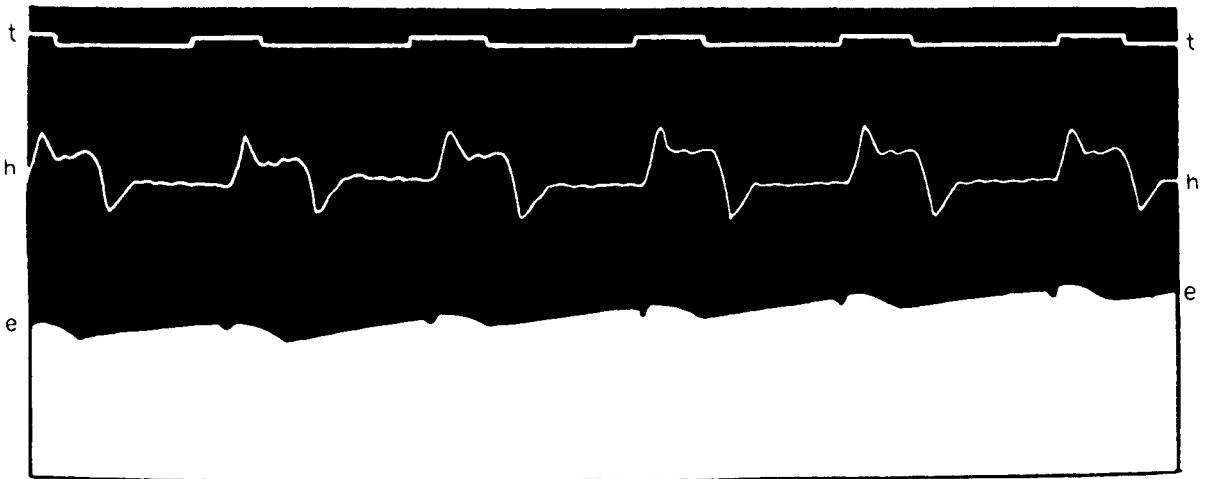


Figure 1.1

The first published electrocardiogram recorded by Waller in 1887. He used the Lippmann Electrometer to record this ECG, which is represented by the black/white interface. The upper tracing represents the time in seconds and the lower tracing represents the movements the chest wall which is synonymous with the apexcardiogram.

1.1.2 Willem Einthoven

In 1901, Willem Einthoven, a Dutch physician from Leiden, was the first to register accurately, and quantitatively, the cardiac electrical current from the body surface by using the string galvanometer (Lamas 1984). The development of the galvanometer was probably initiated by Galvani (Sykes 1987). The string galvanometer was based on the principle that a magnet and a conductor of current will interact. Thus, the galvanometer consisted of a powerful electromagnet between the poles of which was stretched a fine, metallic covered quartz filament. When the connections were completed between the resting subject and the galvanometer, the only significant electrical potentials were those coming from the heart. They were recorded as a deflection of the quartz string. A source of illumination and a system of lenses photographed the string shadow on a moving film.

The string galvanometer was used first in experimental research and eventually was utilized routinely to aid in the clinical evaluation of cardiac diseases. Einthoven in 1906 was also able to develop a method for transmitting the ECG over telephone lines and the methods used together with the results obtained were published in a classic report (see Macfarlane 1989). Only the three bipolar extremity leads, namely, leads I, II and III, were used by Einthoven.

Nevertheless, he was able to detect a variety of

electrocardiographic abnormalities. Later, the concept of an equivalent triangle was introduced by Einthoven and co-workers, by which the body was represented in electrical terms by an equilateral triangle from which the mean QRS axis could also be calculated (Einthoven et al 1913). Einthoven also introduced the terminology of the P, Q, R, S, T components to describe the deflections of the ECG. It was suggested that these letters were selected to leave room for further discoveries such as the U wave, which Einthoven later detected using his string galvanometer (figure 1.2). However, Cooper's (1986) view is that the letters were chosen to conform with the terminology of mathematicians of the day.

1.1.3 Sir Thomas Lewis

Sir Thomas Lewis, a Welsh physician and physiologist contributed to the progress of electrocardiography at the beginning of the 20th century by correlating the ECG with clinical and anatomico-pathologic data. He used the first complete Cambridge electrocardiograph (figure 1.3) and his interests included cardiac arrhythmias and cardiac hypertrophy (Hallman 1981). Lewis published in 1911 the first edition of his book "The Mechanism of the Heart Beat" which according to Hollman (1981) can still be used today without alteration as a first class manual on the principal cardiac arrhythmias. It has excellent illustrations (figure 1.4).

1.1.4 Wilson/Goldberger

In 1932, Wilson and his associates devised the

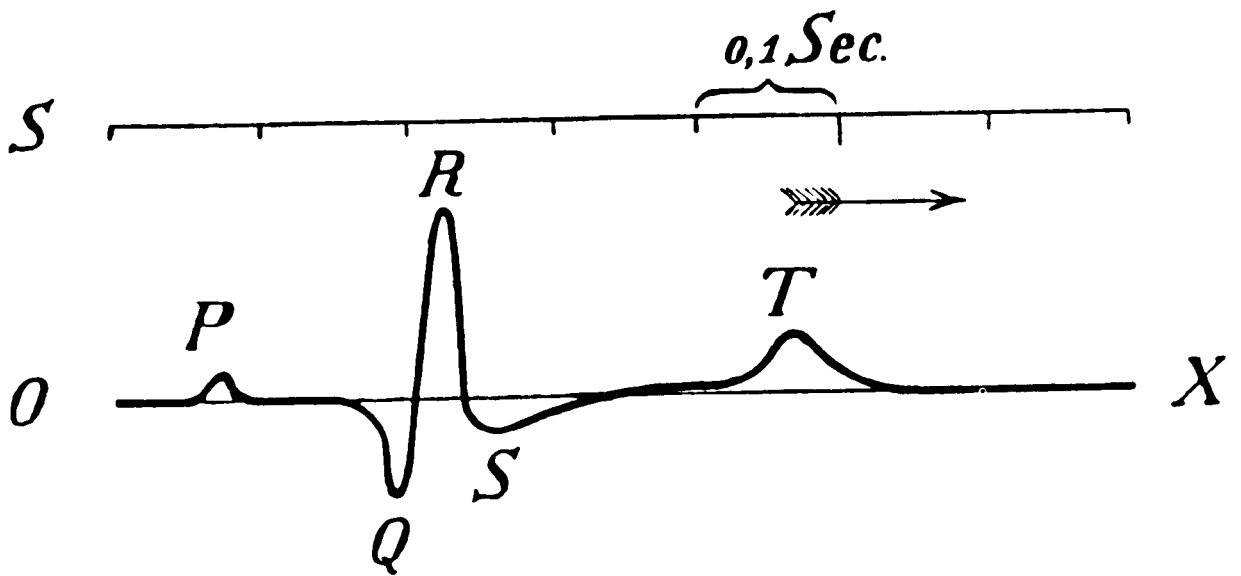


Figure 1.2

The early notation of the electrocardiogram as proposed by Einthoven.

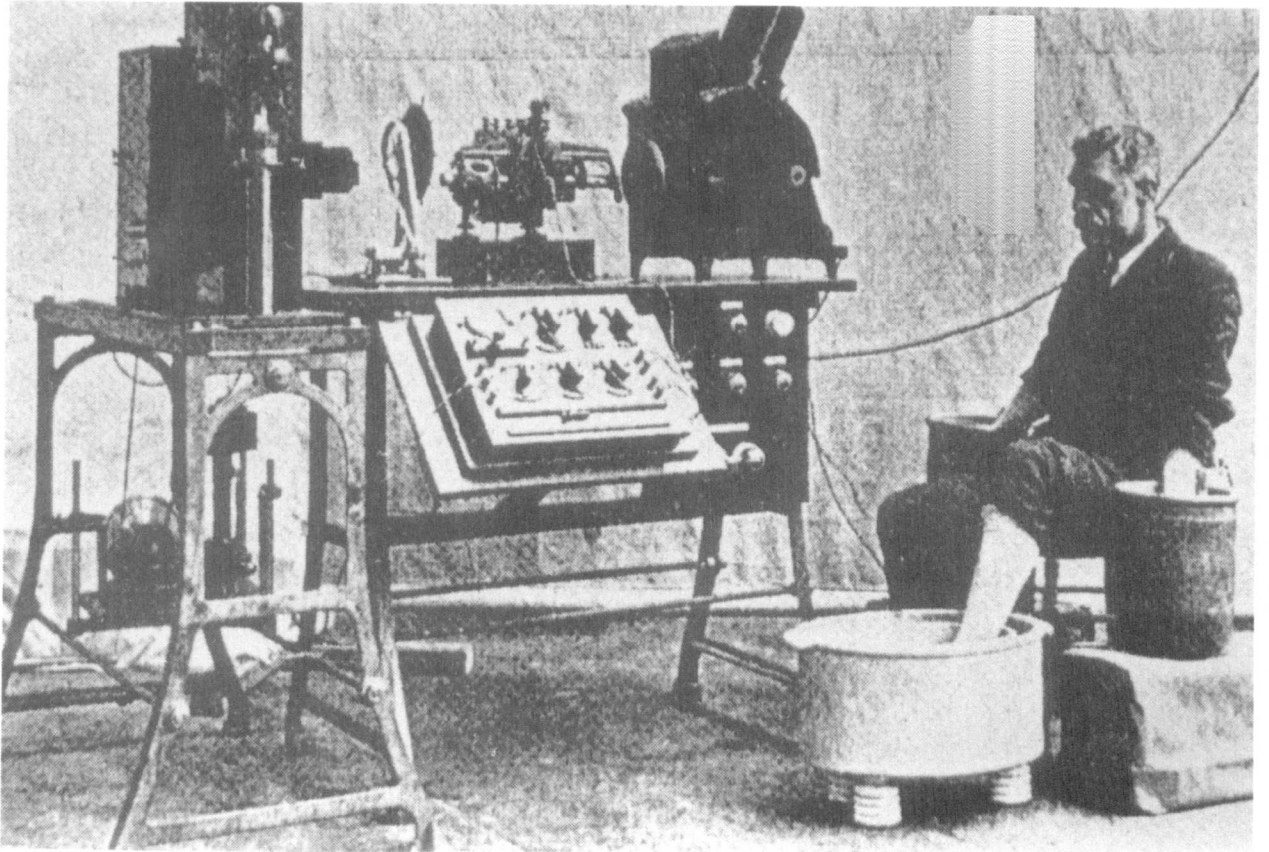


Figure 1.3

A commercial version of an electrocardiograph manufactured by Cambridge Scientific Instrument Company of London in 1911. The galvanometer is at the centre, and to its left is the camera incorporating the plate falling under gravity. The light source at the right side of the galvanometer is needed for recording of the ECG.

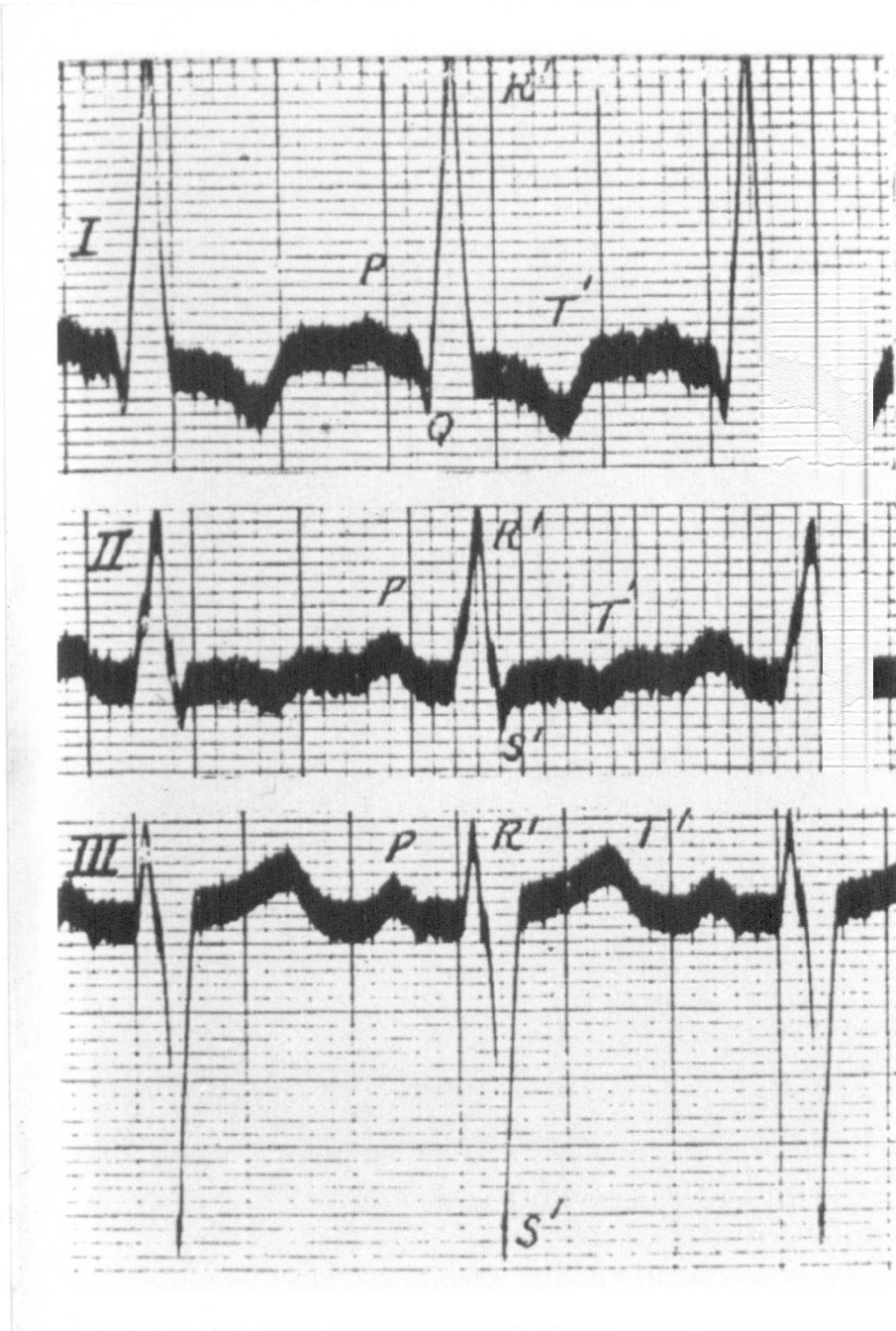


Figure 1.4

An electrocardiogram recorded by Sir Thomas Lewis (leads I, II and III). Lewis thought that this patient had a defect of the right division of the AV bundle, whereas it was later shown that the left division was diseased.

unipolar electrocardiogram (Wilson et al 1932). The unipolar method of recording the electrical activity of the heart was first used for experimental purposes but, like Einthoven's galvanometer, eventually gravitated into the field of clinical medicine. Wilson and colleagues (1932) described the method by which the indifferent electrode was constructed. They linked the left arm, right arm and left leg through equal resistors to a central terminal, which produced a relatively stable reference potential with respect to which the potential at an exploring electrode could be measured. The term unipolar lead was applied because the potential at the central terminal is essentially constant and thus the galvanometer effectively records the variation of potential at a single point. Ultimately, these leads became known as "V" leads so that if the exploring electrode were placed on the left arm, the lead was called "VL".

Kossman and Johnston (1935), later devised six precordial leads, namely, V1-V5 and VE where, in the latter, the electrode was placed at the tip of the ensiform process. The Committee of the American Heart Association (1938) published unilaterally the recommendations of a joint group of Cardiologists from the Cardiac Society of Great Britain and Ireland on one hand and the American Heart Association on the other for the positioning of six precordial leads V1-V6.

Later, Goldberger (1942), completed the American

contribution to the development of the lead system for "conventional" electrocardiography by introducing the so called augmented unipolar limb leads e.g. aVF ($=3/2$ VF).

Thus the scalar ECG of today is composed of 12 leads, namely, the three limb leads I, II and III from Einthoven, three augmented unipolar limb leads aVR, aVL and aVF from Goldberger's modification of Wilson's central terminal, and six precordial leads V1-V6 arising out of Wilson's central terminal.

1.1.5 Vectorcardiography/X,Y,Z leads

A vector is an entity which has a magnitude and a direction. The concept of the vector was introduced into electrocardiography by Waller in 1889 who suggested that the electromotive force of the heart could be represented by a single dipole (Macfarlane 1989). Later, Einthoven et al (1913) introduced the concept of measuring the mean cardiac electrical axis which was represented by a vector. After the introduction of Einthoven's triangle, vectorcardiography started to evolve. Williams (1914) published the first method for deriving a vectorcardiogram from standard limb leads. He calculated the cardiac vector in the frontal plane using the bipolar limb leads introduced by Einthoven. Afterwards, Mann (1920) was able to develop a vectorcardiographic loop and called it a Monocardiogram. Schellong in Germany, Wilson and his team in the United States and, Hollman and Hollman in Germany, independently of each other, developed systems for displaying vectorcardiographic

loops (Macfarlane 1989). Later, Frank (1956) developed his "corrected orthogonal lead system" which is probably the most popular lead system for vectorcardiography wherever the technique is still practised. It recorded three leads X, Y, Z which attempted to measure the component of the resultant cardiac electrical force (vector) in three mutually perpendicular directions - hence the term "orthogonal" lead ECG.

A hybrid lead system was introduced by Macfarlane (1979) to combine the 3 (XYZ) and 12 lead ECG and this is still used in Glasgow Royal Infirmary. Recently, XYZ leads have been derived from the 12 lead ECG (Edenbrandt and Pahlm 1988, Uijen, Van Oosterom, Van Dam 1988, Willems 1984) and from these derived XYZ leads, vectorcardiographic loops can be produced. These derived loops do not compare exactly with the originals but the discrepancies may prove to be of small consequence.

1.2 EVOLUTION OF EQUIPMENT

1.2.1 Computerized interpretation of the electro-cardiogram

Stallman and Pipberger (1961) initiated computerized interpretation of the ECG using the orthogonal lead ECG system, and Caceres and his team (1962) followed using the 12 lead ECG. At that time, large central computers were used for the analysis and at most, leads were recorded in groups of three simultaneously in analogue form. However, Macfarlane and his team in Glasgow Royal Infirmary Cardiology Laboratory among others were able to

advance the technique whereby a small recording unit could be taken to the bedside to make an analogue ECG recording which could subsequently be replayed to a laboratory mini computer for interpretation (Macfarlane et 1972).

Wartak (1970) listed the areas in which computer processing of ECGs may offer advantages to the practising physician including:

- (i) Measurement and analysis of all quantitative aspects of ECGs with an accuracy, rapidity and consistency unattainable by a human being;
 - (ii) Establishment of better diagnostic criteria by analysis of large volumes of ECG data using objective mathematical methods;
 - (iii) Elimination of time-consuming and tedious processing of ECG records by the physician;
 - (iv) Long distance diagnostic processing through common voice-grade telephone networks; and
 - (v) Contribution to clinical research by easy retrieval and analysis of ECG data from various sources.
- Computerized interpretation of the ECG has now expanded from the analysis of the resting ECG to exercise electrocardiography as well as ECG monitoring in the coronary care units.

All the ECGs used in this thesis were recorded by a Siemens Mingorec 4 electrocardiograph or by a locally developed computer compatible electrocardiograph (Watts and Shoat 1987). In both cases, all leads were recorded

simultaneously and sampled 500 times per second. Measurements of the P, QRS and T wave axes, durations and the amplitude of the various deflections of the ECG were made by computer using a locally developed program (Macfarlane et al 1986a). Moreover, every ECG was also scrutinized manually to exclude possible computer errors.

1.2.2 Technical advances in electrocardiography

In addition to computerized interpretation of the electrocardiogram, many advances in the field of electrocardiography have been achieved, including Holter monitoring, and body surface recording of His bundle ECGs.

The use of the microprocessor technology and signal averaging has also led to considerable interest in the study of high frequency components of the ECG. The most frequent application of the signal averaging technique in electrocardiography has been for the detection of ventricular late potentials in patients with ischemic heart disease (Simson and Macfarlane 1989). The late potentials appear to correspond to delayed and fragmented ventricular activation which can be observed with direct ECG recordings in patients with ventricular tachycardia (Simson and Macfarlane 1989). Signal averaging is also applied in the recording of the His bundle activity from the surface of the body (Berbari et al 1973, Hashimoto and Sasayama 1975, Takeda et al 1979). Current research into physical principles and clinical applications may

well lead to other new horizons.

1.2.3 Invasive investigation of the cardiovascular system

Claude Bernard catheterized both the right and left ventricles of a horse by means of a retrograde approach from the jugular vein and carotid artery (Cournand 1975). However, Werner Forssmann was the first to pass a catheter into the heart of a living human being (Grossman 1985). At the age of 25, he exposed a vein in his left arm, introduced a ureteral catheter into the venous system, and advanced it under fluoroscopic control into the right atrium. He then walked to the Radiology Department of the hospital, where the position of the catheter was confirmed by a chest x-ray. During the next two years, Forssmann continued to perform catheterization studies, including six additional attempts to catheterize himself

Klein in 1930, reported on cardiac catheterization of the right ventricle in 11 patients on whom measurements of cardiac output were made by using the Fick principle (Grossman 1985). However, Cournand and Ranges (1941), made a remarkable series of right sided cardiac catheterizations in humans.

Left sided cardiac catheterization was first introduced by Zimmerman and colleagues in 1950 (Grossman 1985). Later, Seldinger (1953) developed the percutaneous technique which soon was applied to cardiac catheterization of the right and left hearts. Retrograde

left sided cardiac catheterization and contrast left ventriculographic angiography allowed investigators such as Arvidsson (1958), Chapman et al (1958) and Dodge et al (1960) to utilize two dimensional radiographic techniques for the opacification and determination of left ventricular volume according to area-length measurements. The contrast ventriculographically estimated left ventricular volumes were later used as the gold standard for the calculation of left ventricular volumes from the M-mode echocardiogram (Pombo et al 1971).

Radner (1945) was the first to outline the coronary arteries in man. The earliest studies consisted of incidental opacifications of the coronary arteries at the time of retrograde aortography. Sones and co-workers reported a straightforward approach to achieve opacification of the coronary arteries by which deliberate and selective catheterization of each vessel was possible (Sones et al 1959). In 1967, the percutaneous transfemoral approach for coronary arteriography was introduced by Judkins (Grossman 1985). The introduction of coronary arteriography allowed a wider and more precise evaluation of electrocardiographic features of coronary artery disease, whereas prior to this technique, postmortem data provided the only comparative material against which the ECG signs of coronary artery disease could be studied.

1.2.4 Echocardiography

Echo sounding is a technique used by certain birds

and animals for distance perception. The first application of echo sounding by a human was in the 1920's for depth recordings in oceanographic studies and submarine detection. The principles of diagnostic ultrasound have their roots in navy sonar, which uses sound impulses to detect objects and measure distance in water (Felner 1986). Ultrasound is defined as a sound above the threshold of human audibility (20,000 cycles per second (c/s) or Hertz (Hz)). Ultrasonics, the technology of high frequency sound waves deals with the transmission of these high frequency or pressure waves through a medium. The high frequency vibrations are created by an appropriate piezo-electric crystal (transducer) fed with alternating electric current. A short burst or pulse of high frequency low intensity sound is emitted from the transducer and directed through the human body to detect boundaries between structures of different acoustic impedance. The depth and position of the different sonic waves (echoes) returned from inside the body can be plotted and recorded. Cardiac ultrasound or echocardiography is the transmission of the pulsed ultrasound through the heart and the detection of the returning echoes detailing the position and movement of the cardiac acoustic interface.

Clinical echocardiography requires ultra-high frequency sounds of 2-5 MHz. Sound of this frequency can be transmitted on a narrow beam and directed along a rather well defined path through the soft tissues of the

body with a transmission speed of 1540 meters/second. The basic circuitry of the pulsed reflected ultrasound system causes the piezo-electric crystal to alternate extremely rapidly between functioning as a transducer and as a receiver of ultrasound. During the transmission cycle, the electronic cycle provides a very short burst or pulse (500-1500 pulses per second) of high frequency waves. During the much longer receiving cycle, the piezo-electric crystal functions as a receiver which detects ultrasound vibrations, and transforms them back into an electric signal that can be amplified, appropriately displayed and recorded. The transducer acts as a transmitter for less than 1% of its operation and hence as a receiver for more than 99%. Thus the amount of ultrasound energy passing through the tissues is minimal.

Keidel in 1950 was one of the first investigators to use ultrasound to examine the heart (Felner 1986). However, it was not until the mid 1950's that Edler and Hertz pioneered the use of pulsed ultrasonic techniques in the description of certain aspects of cardiac anatomy (Felner 1986). Thereafter, echocardiography was popularized in the United States by Holmes and colleagues (Winters 1984). Initially, echocardiography was used for assessment of mitral stenosis by Edler and Gusterson (Felner 1986), then for pericardial effusion (Feigenbaum et al 1966) and assessment of cardiac chambers by Jogner in 1957 (Winters 1984). Applications of echocardiography

extended to the calculation of left ventricular volumes (Pombo et al 1971) and masses (Troy et al 1971, Devereux and Reichel 1977). In this thesis, echocardiography is used to assess and classify left ventricular hypertrophy (LVH) and enlargement as the gold standard for evaluation of electrocardiographic criteria for LVH/enlargement.

1.2.5 Nuclear cardiology

The use of radionuclide tracers to study cardiovascular dynamics was reported initially by Blumgart and Weiss (1927). These workers used radon gas injected intravenously and employed a modified cloud chamber to measure circulation time in humans. Prinzmetal and colleagues in 1949 described the gross characteristics of the first pass radiocardiogram using Sodium 22 and a Geiger-Muller counter positioned over the precordium (Zaret and Benger 1986). Bender and Blum in 1963 introduced the autofluoroscope which is a multicrystal scintillation camera (Zaret and Benger 1986). Anger and colleagues (1965) were able to define the cardiac transit with analogue images obtained from a prototype single-crystal scintillation camera. Mullins et al (1969), described a method for recording end-diastolic and end-systolic volumes of the heart. In the early 1970's, the concept of the electrocardiographic gating of the equilibrium cardiac blood pools was proposed as a means of evaluating regional wall motion (Zaret et al 1971) and left ventricular ejection fraction (Strauss et al 1971). However, the calculation of right

ventricular ejection fractions by radionuclides was achieved in the early 1980's (Slutsky et al 1980, Hollman et al 1981). Donato in 1973 analysed the quantitative aspects of the radiocardiogram using a newly developed shielded and collimated scintillation probe (Zaret and Benger 1986). This allowed definition both of cardiac dynamics and myocardial blood flow. Later, computer techniques were applied to the equilibrated cardiac blood pool and first transit studies (Burow et al 1977) and were followed by the addition of exercise stress testing to the assessment of ventricular performance (Borer et al 1977).

The application of radionuclides in evaluation of myocardial perfusion in man was first introduced by Carr and associates in 1963 (Zaret and Benger 1986). They used radioactive Caesium as a potassium analogue in the diagnosis of myocardial infarction. Later, Thallium 201 was introduced as a potassium analogue by Lebowitz and associates in 1973 (Zaret and Benger 1986). Sharpe et al (1978) reported that myocardial imaging with Technetium 99 Pyrophosphate provided more direct evidence of acute infarction involving the right ventricle.

In this thesis, the radionuclide angiographically determined ejection fraction of the left ventricle as well as perfusion studies are used as gold standards for studying LV function predicted from the ECG.

2. EVOLUTION OF ECG CRITERIA AND AIMS OF THE STUDY

A major aim of this thesis is the study and development of ECG criteria particularly for application by computer methods. For this reason, conventional scalar ECGs require to be correlated with other echocardiographic, contrast angiographic and radionuclide data. In particular, this study will concentrate on patients with left ventricular hypertrophy and ischemic heart disease. For this reason, the evolution of autopsy and ECG criteria of LVH, as well as of ECG criteria in ischemic heart disease, are reviewed.

2.1 LEFT VENTRICULAR HYPERTROPHY

2.1.1 Autopsy diagnosis of left ventricular hypertrophy

Muller, in 1883, undertook the first study on autopsy materials for anatomico-pathologic diagnosis of left ventricular hypertrophy (LVH) (Bove, Rowlands and Scott, 1966). However, his findings were not supported by antemortem systemic arterial blood pressures. Zeek (1942) formulated weight criteria for normal and hypertrophied hearts including left and right chambers, and compared them with antemortem systemic blood pressures. Bove et al (1966), introduced the chamber partition technique for identification of the left ventricle at autopsy. This is a more reliable method for determining the weight of the left ventricle. Dower, Horn and Ziegler (1967) tried to find other simpler, reliable equations for autopsy diagnosis of LVH thus avoiding the

meticulous chamber partition technique introduced by Bove et al (1966). They concluded that the ratio of the weight of the heart in grams to the length of the cadaver in centimeters, could be used so that a ratio in excess of 2.26 for males, or 2.06 for females indicated autopsy LVH. The autopsy based weight criteria of LVH formed the cornerstone for subsequent studies of ECG-LVH to the extent that validation of most existing ECG criteria, with the notable exception of the study of Casale et al (1985), has been with respect to the weight of the heart at autopsy. Only in the mid 1980s, did echocardiography provide another gold standard against which ECG-LVH criteria could be evaluated and developed.

2.1.2 Evolution of voltage criteria of ECG-LVH

Increased QRS voltage is the earliest criterion related to autopsy LVH. An index of R and S voltages in the limb leads I and III, i.e. $(RI - RIII) + (SIII - SI)$ was introduced by Lewis (1914). When the index, later known as the Lewis index, was $+1.7$ mV or more, ECG LVH was diagnosed. Hermann and Wilson (1922) through ECG-postmortem correlations, confirmed that the Lewis index was useful in the diagnosis of ECG-LVH. Many other limb lead voltage criteria were introduced, namely;

- (i) $R I + S III > 2.5$ mV; $R I > 1.5$ mV (Gubner and Ungerleider 1943),
- (ii) $R I > 1.3$ mV (Manning and Smiley 1964),
- (iii) $R aVL > 0.75$ mV (Mazzolini et al 1964),
- (iv) $R aVL > 1.2$ mV, $R aVF > 1.3$ mV or $S aVR > 1.4$ mV

(Schack, Rosenman and Katz 1950).

(v) $R_{aVL} > 1.3 \text{ mV}$ or $R_{aVF} > 2.0 \text{ mV}$ (Goldberger 1949)

(vi) $R_{aVL} > 1.1 \text{ mV}$ or $R_{aVF} > 2.0 \text{ mV}$ (Sokolow and Lyon 1949).

The precordial voltage criteria for the diagnosis of ECG LVH were introduced by Wilson et al (1944) who suggested that $S_{V1} > 2.4 \text{ mV}$ reflected autopsy LVH. Later, other precordial voltage criteria were introduced for the diagnosis of ECG-LVH. These include;

- (i) the sum of the greatest R and the greatest S in the precordial leads $> 4.0 \text{ mV}$ (McPhie 1958),
- (ii) $(S_{V1} \text{ or } S_{V2}) + R_{V6} > 4.0 \text{ mV}$; the sum of R and S in any single precordial lead $> 3.5 \text{ mV}$ (Grant 1957),
- (iii) $S_{V1} + (R_{V5} \text{ or } R_{V6}) \geq 3.5 \text{ mV}$ (Sokolow and Lyon 1949). This criterion has become the most widely used in clinical practice.

2.1.3 Evolution of non-voltage criteria of ECG-LVH

Left axis deviation (LAD) of -30 to -90 degrees was the first non-voltage criterion of ECG-LVH. It was introduced by Gubner and Ungerleider (1943) and later confirmed by Grant (1956). However, the onset of the intrinsicoid deflection in $V5$ or $V6 > 0.05$ seconds as an ECG sign of LVH was suggested by Noth et al (1947). Morris et al (1964), pointed out that the left atrial involvement seen on the ECG as a negative P terminal force in $V1$ is a useful criterion of ECG-LVH. Carter and Estes (1964) showed that in addition to LAD as well as the voltage in the limb and precordial leads, asymmetric

ST segment depression and T wave inversion in leads V5 and V6, and prolonged QRS duration correlated significantly with LVH at autopsy. The appearance of these non-voltage criteria of ECG-LVH, led to the emergence of a combination of voltage and non-voltage criteria.

2.1.4 Combination of voltage and non-voltage criteria in ECG-LVH

Romhilt and Estes (1968) introduced a point score system for the diagnosis of ECG-LVH (for details see chapter 8), and this was based on ECG-autopsy correlations. The point score system of Romhilt and Estes was modified by Macfarlane (1987) and was used routinely in our laboratory until further modified as discussed in Chapter 8. The so called Cornell criteria introduced by Casale et al (1985), used the combined amplitude of R aVL and S V3 together with the amplitude of the T wave in lead V1, with indexation to age and sex. This group used echocardiographically defined left ventricular masses as their gold standard as they had been shown to correlate well with the weight of the left ventricle at necropsy (Troy et al 1972, Devereux and Reichek 1977). This approach provided another mechanism via which ECG-LVH criteria could be developed from echocardiographic LV masses thereby avoiding meticulous dissection of the heart at autopsy.

2.1.5 Left ventricular hypertrophy/enlargement

Pathophysiologically, LVH is of two types, namely

concentric and eccentric (Braunwald 1980, Oparil 1985, Ford 1976, DePace et al 1983, Sasayama et al 1976, Ross 1974, Graham et al 1974). In both types, the left ventricular mass is increased, but while the left ventricular volume is normal or reduced in the former, it is increased in the latter (figs. 2.1 and 2.2). According to the Brody effect (1956), increased intraventricular volume causes potentiation of QRS voltages. In support of the Brody effect, Antman et al (1979) showed that LVH due to left ventricular dilatation (e.g.. due to aortic regurgitation), causes greater potentiation of QRS voltages than does a thickened myocardium due to aortic stenosis. However, in contradiction to Antman et al (1979), Devereux et al (1983), found that for a given left ventricular mass, ECG voltage criteria of LVH are independent of LV dilatation or other geometric variables. Therefore, to date, no ECG criteria have evolved by which concentric and eccentric types of LVH can be differentiated from each other.

2.1.6 Limitations of ECG-LVH criteria

More than twenty criteria have been designed for the diagnosis of ECG-LVH (Romhilt et al 1969). Unfortunately, these criteria, like most other criteria, have an inverse relationship with respect to specificity and sensitivity. Allenstein and Mori (1960), in a review of ECG-LVH, concluded that in general those criteria with a high degree of specificity have low sensitivity and those with low specificity have high sensitivity. This is clearly an

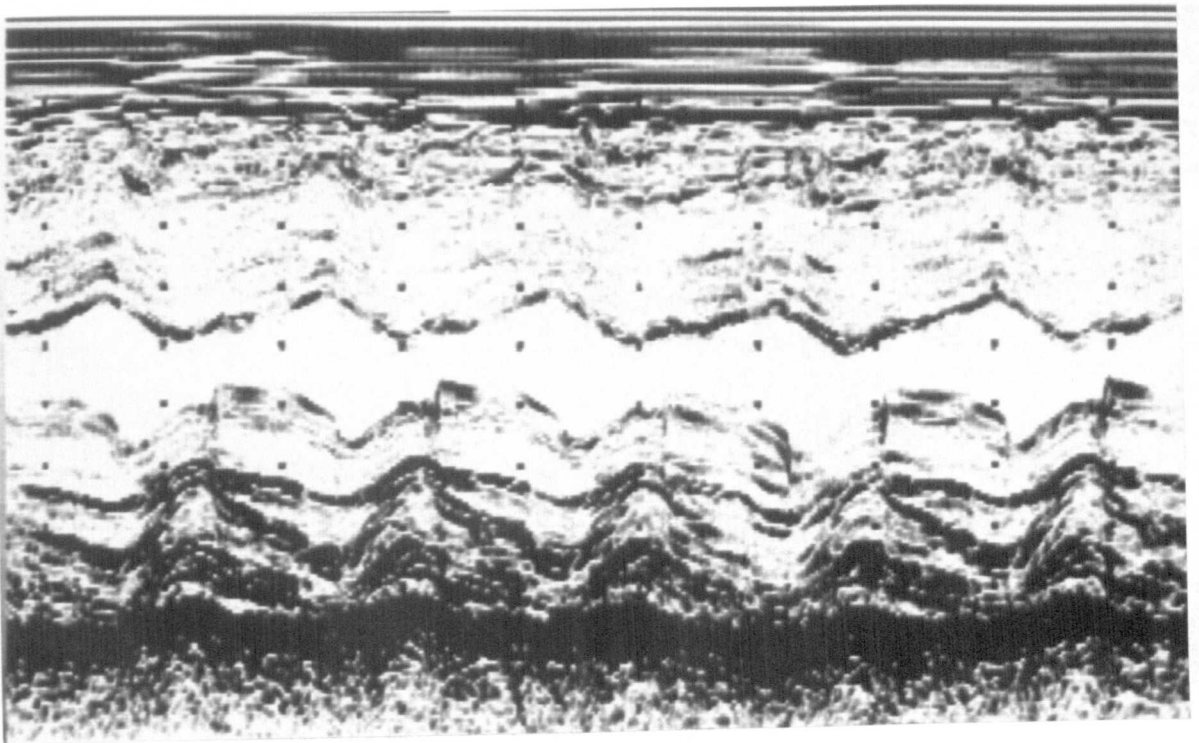


Figure 2.1

M-mode echocardiogram of a patient with concentric left ventricular hypertrophy. It was recorded by a Diasonic Cardiovue 3400 Phased Array imaging system. It shows that the interventricular septum and the posterior wall thickness of the left ventricle are greatly increased at the expense of the left ventricular cavity size. The resultant left ventricular hypertrophy is concentric because the left ventricle is not dilated.

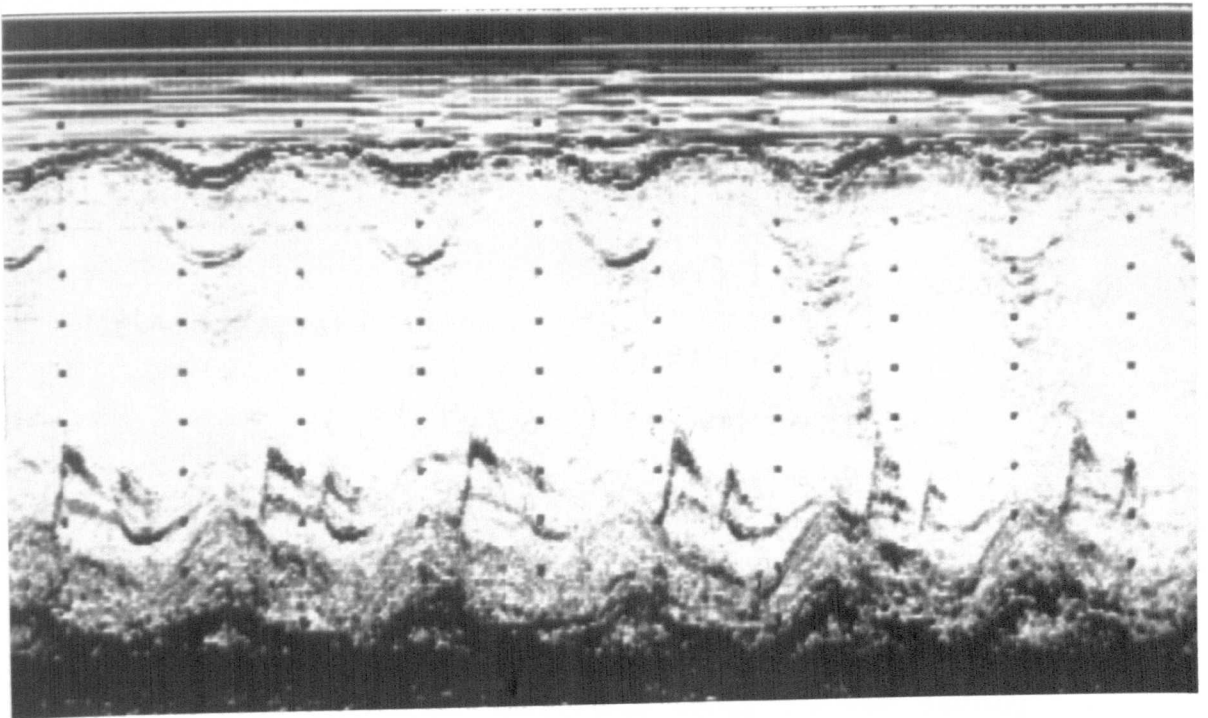


Figure 2.2

M-mode echocardiogram of a patient with eccentric left ventricular hypertrophy. It was recorded by a Diasonic 4 Cardiovue 3400 Phased Array imaging system. It shows that the left ventricular internal dimension is increased while the interventricular septum and the posterior wall of the left ventricle have normal thickness. The patient has an eccentric left ventricular hypertrophy as the volume and mass are increased beyond the normal limits.

indication that better criteria are required, while at the same time, the large number of ECG criteria published indicates the difficulties encountered in this field. In general, all the ECG criteria for LVH with the exception of the Cornell criteria (Casale 1985) have evolved from ECG-autopsy correlations. This produces the following defects:

- (i) LVH diagnosed at autopsy deals with the most extreme degrees of LVH. ECG criteria derived from this material are probably not applicable to the total population of LVH during life which includes mild to moderately severe LVH as well as those types of LVH which may be reversible.
- (ii) No voltage criteria from ECG-autopsy correlations have been indexed to the age and sex of the subjects studied. It is well known that QRS voltage decreases with age (Manning and Smiley 1964, Macfarlane and Lawrie 1989a) and that women have lower QRS voltage than men (Simonson 1961). They also vary with race (Chen, Chiang and Macfarlane 1989).
- (iii) All ECG-LVH criteria including the Cornell criteria (Casale et al 1985) have been based on left ventricular mass, and therefore the role of left ventricular volume in ECG-LVH remains unclarified, with or without the effects of mass.
- (iv) Myocardial infarction (whether acute or chronic, single or multiple), affects QRS voltage by

decreasing the amplitude of the R wave and/or through the development of new Q waves. While, anteroseptal myocardial infarction (MI) causes Q waves in leads V1, V2 and V3, it is not rare to find pathological Q waves in the same leads in patients with LVH but no MI (Chou 1986a). Moreover, LVH and anteroseptal MI frequently co-exist. Therefore the criteria for the diagnosis of ECG-LVH in the presence of pathological Q waves in leads V1, V2 and V3 need to be clarified, especially so because others have found that MI generally increases sensitivity and decreases specificity of ECG-LVH (Murphy et al 1984).

2.2 ELECTROCARDIOGRAPHIC CRITERIA IN ISCHEMIC HEART DISEASE

The electrocardiographic criteria of myocardial ischemia and infarction have evolved from autopsy studies. These criteria have limitations because the data obtained at postmortem do not represent the full spectrum of ECG characteristics of coronary artery disease. However, with the relatively recent advent of radionuclide and contrast angiographic studies of the cardiovascular system, it is now possible to re-evaluate these criteria.

Pardee (1925) was the first to describe the symmetrical T wave inversion of myocardial infarction. Wilson in 1931 pointed out that the coronary T wave of

myocardial ischemia and infarction was due to prolongation of the action potentials in the regions of the ventricle immediately adjoining the area of the infarction (Lipman and Massie 1969b). T-wave inversion simulating myocardial ischemia has also been described in healthy individuals as a normal variant (Thomas et al 1960, Chou 1986b, Lichtman et al 1973, Hane-Paparo et al 1971, Gottlieb et al 1975). The morphologic similarities of normal T wave inversion with those due to symptomatic coronary artery disease, led some authors to utilize other criteria in their differentiation, such as the angle between the QRS axis and the T wave axis (Schamroth 1976, Rowlands 1980).

Prinzmetal et al (1954) as well as Shaw et al (1954) confirmed that myocardial infarction causes diminution of the R wave and/or appearance of Q waves. Further research attempted to correlate the extent of the myocardial necrosis and the changes of the QRS complex. Experimental (Shaw et al 1954, Hillis et al 1976) as well as clinical observations (Askenazi et al 1977, Hardarson, Henning and O'Rourke 1976, Selvester et al 1972), demonstrated a relationship between the changes of the QRS complex and the extent of myocardial infarction. The work of Awan et al (1976), suggested that the results of precordial mapping of Q waves in patients with acute anterior myocardial infarction correlated well with the extent of the necrosis assessed with measurements of left ventricular dyskinesia and ejection fraction. This

ultimately led Selvester and his colleagues (1989) through a series of experimental and clinical studies, to formulate a QRS point scoring system for infarct sizing. They also suggested its use for calculation of the left ventricular ejection fraction in patients with myocardial infarction (MI) as discussed in detail in chapter 3.

2.3 AIMS OF THE STUDY

2.3.1 Development of ECG criteria on the basis of modern investigative techniques

Now that echocardiography, cardiac and coronary catheterization, and nuclear cardiology facilities are available in parallel with computerized electrocardiographic measurements, it is possible to re-evaluate existing ECG criteria and develop new criteria. This can be undertaken by correlating computerized 12 lead ECG measurements with anatomical, physiological and pathological data obtained from patients with, for example myocardial ischemia, myocardial infarction, or left ventricular hypertrophy. The purposes of the study can be summarized as follows:

- I. Assessment of left ventricular function in patients with myocardial infarction (acute and old) based on criteria of infarct sizing derived from the scalar ECG.
- II. Improvement of interpretation of repolarization wave abnormalities of myocardial ischemia in the absence of pathological Q waves.
- III. A complete evaluation of the ECG diagnosis of left

ventricular hypertrophy in its different forms and particularly, a study of the possibility of differentiating hypertrophy from enlargement.

2.3.2 Myocardial ischemia and infarction: diagnosis and assessment of left ventricular function

The evaluation of left ventricular function and the prediction of LV ejection fraction by Selvester's QRS point scoring system in patients with acute MI, is still in its infancy and needs more evaluation and possible modifications before it becomes well accepted. The whole idea is principally based on the concept that the QRS voltage and left ventricular ejection fraction are functions of the viable myocardium. Moreover, how precisely accurate the performance of Selvester's QRS scoring system is with respect to the following is unknown; (i) evaluation of left ventricular function in the immediate post-infarct period and three months after myocardial infarction, and (ii) exact prediction of left ventricular ejection fraction in individual patients.

Separately, a high P terminal force (PTF) in lead V1 has been shown to correlate well with raised pulmonary capillary wedge pressure in patients with acute myocardial infarction (Heikkila 1973, Chandraranta 1978). However, no study has correlated the PTF in V1 with left ventricular ejection fraction in patients in the immediate post-infarct period or with old MI. LV function of patients with acute MI in the immediate post-infarct period and three months after acute MI can be studied

respectively by left ventricular ejection fraction obtained with Tc-99 and contrast LV gram ejection fractions.

Isolated repolarization wave abnormalities are recognized features of symptomatic coronary artery disease (CAD). However, T wave inversion in the precordial leads and occasionally in the inferior leads, may occur as a normal variant in healthy individuals (Thomas et al 1960, Chou 1986b, Hane-Paparo et al 1971, Lichtman et al 1973, Gottlieb et al 1975). It is also well known that pre-menopausal women may exhibit T wave changes in the inferior leads. Therefore, the significance of isolated T inversion, whether in the precordial or inferior leads and in particular lead aVF, in relation to symptomatic CAD needs investigation. Moreover, there is controversy according to the opinions of different authors over the role of the QRS-T angle in the differentiation of normal and abnormal T wave inversion.

2.3.3 Electrocardiography in LVH/enlargement

Left ventricular masses obtained from M-mode echocardiograms can be utilized for studying the ECG criteria of left ventricular hypertrophy. Moreover, from the ECG standpoint, there have been few if any studies which have tried to develop criteria for separating increased muscle mass from increased mass and volume. Cabrera introduced the terms systolic and diastolic overload to describe LVH with and without T wave

inversion, but no echo correlations were involved at that time (Cabrera and Monroy 1952). For these reasons it was decided to study the ECG parameters which might help to separate LVH due to pressure and volume overload, and also to evaluate the electrocardiographic concepts of systolic and diastolic overload. For the above mentioned reasons, an echocardiographic study was thought to be essential for the classification of LVH into concentric and eccentric types.

However, an echo classification of LVH/enlargement can be affected by the particular convention used for the measurement of the LV dimensions. Two different echocardiographic conventions are commonly used, namely the recommendations of the American Society of Echocardiographers (Sahn et al 1978) for the estimation of LV volume (Pombo et al 1971) and mass (Troy et al 1972) while, the so called Penn convention has been used for the calculation of LV mass (Devereux and Reichek 1977). The LV mass estimated by the Penn convention is significantly lower than the mass calculated utilizing the method described by Troy et al (Savage 1987). In the Penn convention, the endocardial echoes are excluded from the measurements of the interventricular septum and the free wall of the ventricle, thereby causing the internal dimension to be higher than when it is measured according to the recommendations of the American Society of Echocardiographers (ASE). Therefore, it would be expected that a larger Penn than ASE derived left

ventricular volume would be obtained for the same individual. When the effects of echocardiographically determined LV mass and volume on ECG-LVH criteria are to be studied, a decision has to be made whether to use the Penn convention for the mass and ASE for the volume, or to use regression equations converting volume from Penn to the ASE convention, because the cut-off point of left ventricular volume of 90 ml/m^2 as an indication of left ventricular dilatation has been based on ASE measurements.

An echocardiographic index called the relative wall thickness which relates the thickness of the free wall to the internal radius of the LV, has been suggested as useful in classifying LVH in a sample of the general population (Savage et al 1987). This approach resulted in defining a group of patients with increased LV mass and normal LV internal dimension. These authors referred to this group as having eccentric non-dilated LVH. However, pathophysiologically, an increased LV volume is the stimulus for eccentric LVH. Therefore, it was not clear at the outset how accurately the relative wall thickness can separate the various types of LVH/enlargement when LV volume and mass are used as a gold standard, especially so in a sample of a cardiac population where a wide variety of left ventricular geometry is expected. For this reason, it was decided to study a large sample of cardiac patients with adequate M-mode echocardiograms, from which relative wall thickness and indexed left

ventricular volume and mass could be derived.

In view of the availability of methods (i) to calculate left ventricular masses and volumes by echocardiography, and hence for classification of LVH/enlargement, and (ii) for computerized measurement of the 12 lead ECG, a study of the following aspects of the ECG diagnosis of LVH was designed:

- (1) The comparative value of the commonly used ECG-LVH criteria and the newly introduced Cornell criteria, which involve the amplitude of the T wave in V1, the R wave in aVL and the S wave in V3.
- (2) The ECG parameters that correlate independently with left ventricular mass and which might therefore be used for improvement of ECG-LVH criteria.
- (3) The possibility of developing a regression equation involving ECG parameters for estimation of LV mass. The question of whether the various types of LVH require different equations also required to be investigated.
- (4) The distribution of the voltage and non-voltage criteria of ECG-LVH according to the echocardiographically determined degrees of severity.
- (5) The detection of the ECG parameters that may possibly differentiate LVH and LV enlargement.
- (6) The evaluation of the concept of systolic and diastolic overload of the ECG-LVH introduced by Cabrera and Monroy (1952).
- (7) How the presence of pathological Q waves in leads V1

V2 and V3, affects the diagnosis of ECG-LVH. All the patients in this group require to be carefully assessed for MI by clinical history, cardiac enzymes and possibly also by coronary catheterization when the latter is indicated for other reasons.

3. MYOCARDIAL INFARCTION: EVALUATION OF LEFT VENTRICULAR FUNCTION FROM THE SCALAR ELECTROCARDIOGRAM

3.1 INTRODUCTION

Left ventricular end-diastolic pressure (LVEDP) rises in the majority of patients with acute myocardial infarction (acute MI) even when uncomplicated (Hunt et al 1970, Hamosh and Cohn 1971, Karlinger and Ross 1971, Rahimatoola et al 1971). When the acute MI is transmural, LV dysfunction occurs in every patient with consequent rise of left ventricular filling pressure and this may be followed by pulmonary oedema (Heikkila et al 1971). The scalar electrocardiogram (ECG) has previously been utilized to predict LV function following acute MI through two main approaches, namely (i) by analysing the P terminal force in lead V1, and (ii) by using Selvester's QRS scoring system (Selvester 1989).

The P terminal force in lead V1 (PTFV1) has been defined as the product of the amplitude and duration of the negative terminal deflection of the P wave in lead V1 (Morris et al 1964). Heikkila et al (1973) found that a $PTFV1 \geq 3$ millivolt.msec (mV.msec) correlated highly with a pulmonary capillary wedge pressure (PCWP) ≥ 12 mm Hg in patients with acute MI. This was also confirmed by Chandraranta and Hodge (1978). However, the value of the PTFV1 in patients with acute MI has not been studied in relation to the left ventricular ejection fraction, especially when the latter represents the global LV

function which can be quantitated non-invasively both by echocardiography and radio-isotope scans.

A QRS scoring system (37 criteria/29 points) for infarct sizing based on computer simulation of the sequence of ventricular activation has been developed by Selvester and his associates (Selvester et al 1965, Selvester et al 1967, Selvester et al 1968). An extended QRS scoring system (54 criteria/32 points) was found to correlate better with infarct size at postmortem than the original system (Selvester, Sanmarco, Solomon and Wagner 1982, Selvester, Solomon and Wagner 1982). However, the 54 criteria/32 points scoring system which permits assessment of left ventricular function has not been widely studied in acute MI. It remains unclear whether the QRS scoring system can predict, with a reasonable degree of accuracy, the ejection fraction (EF) in individual patients with acute MI. The effect of the site of the infarct on the prediction of EF and on the 54 criteria/32 points scoring system also needs to be determined.

For these reasons, it was decided to study Technetium 99 (Tc 99) derived ejection fractions in relation to the PTFV1 and the 54 criteria/32 points scoring system in patients with acute myocardial infarction (acute MI). Moreover, impaired left ventricular function is an important factor in predicting survival after myocardial infarction. Adverse prognosis has been demonstrated after MI in patients with a low ejection fraction

(Hammermeister 1979a). The multicentre post-infarction research group (1983) reported that patients with a high global ejection fraction had a better one year survival after myocardial infarction independent of the extent of coronary artery disease. However, the correlation of left ventricular ejection fraction estimated from contrast left ventriculograms with Selvester's QRS scoring system and/or the PTFV1 in patients with old MI remains to be clarified. For this reason, left ventricular ejection fractions of a group of patients with an MI more than 3 months old were also studied with respect to Selvester's QRS scoring system and the PTFV1.

3.2 PATIENTS AND METHODS

3.2.1 Acute myocardial infarction

Consecutive patients who had chest pain suggestive of cardiac origin or who were considered clinically to have suffered an acute MI were admitted to the coronary care unit and included in the study. All patients were initially considered regardless of age, sex and co-existent medical conditions, but some were excluded subsequently on account of major cardiovascular complications. Furthermore, patients with clinical problems that might affect the P wave were excluded, e.g. aortic and mitral valve disease, cor pulmonale, supraventricular arrhythmias or atrial fibrillation. Patients with electrocardiographic left ventricular hypertrophy were also excluded from the evaluation of the PTFV1 as the latter is known to be related to hypertrophy

of the LV (Romhilt and Scott 1972). Diagnosis of acute MI was made retrospectively in accordance with W.H.O. criteria (W.H.O. Report 1971) which are based on three daily serial ECGs and routine enzymes.

Blood samples for creatine phosphokinase (CPK) were taken 4 hourly up to 30 hours (maximum 7 samples). ECGs were recorded on a Siemens Mingorec 4, or on a locally developed computer compatible electrocardiograph (Watts and Shoat 1987). ECGs were analysed by a computer program (Macfarlane et al 1984) which allowed accurate measurements of amplitude and duration of ECG waves. The ECGs were also checked manually to detect notches in the QRS complex and to exclude possible computer errors. All ECGs were recorded at a speed of 25 mm/sec. and a sensitivity of one millivolt per centimeter. A PTFV1 \geq 4.0 mV.msec is considered as abnormal in our laboratory. The PTFV1, and QRS score based on the 54 criteria/32 point system (figure 3.1), were calculated for each ECG on the 3rd day post MI. The diagnosis of acute MI was confirmed by Thallium 201 scans obtained both in the 45 degree left anterior oblique and anteroposterior projections on admission. These were repeated after approximately 24 hours. Radionuclide EFs from Tc 99 scans were calculated on the 3rd day post infarct and were converted to predicted contrast left ventriculographic (LV gram) EFs by a correction factor used in our laboratory (Hutton 1987):

$$\text{Predicted LV gram EF} = (\text{Radionuclide EF} \times 1.2) + 6$$

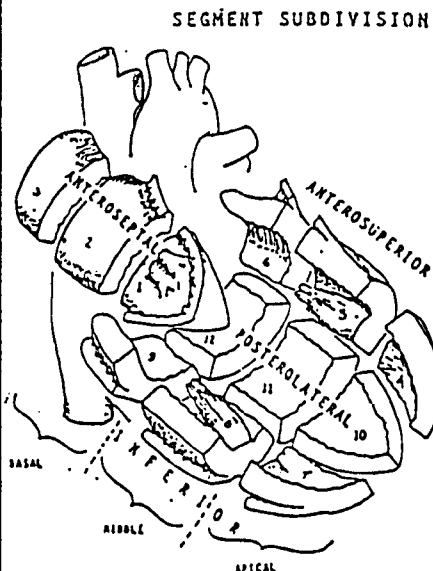
PANEL A

PAKEL B

[illegible]

INFARCT SIZE - ECG SCORE - HOW TO - -

1. CIRCLE EACH ECG CRITERIA MET FROM... PANEL A
(If more than 1 criteria in bracket
met circle only 1 with most points)
2. CIRCLE POINTS SCORED EACH CRITERIA.. PANEL A
3. CIRCLE STV INFARCT IN EACH SEGMENT
FOR EACH QRS CRITERIA MET..... PANEL B
4. PLACE ⊙ (FOR EACH 1% LVI) FROM..... PANEL B
IN PROPER SEGMENT IN DIAGRAM BELOW



INFARCT SIZE: EJECTION FRACTION:

TOT. ECG PTS.X3 = 2LY INFARCTED (2LYVI)

65-2LYI - ESTIMATED EJ. FR. (EEF)

INFARCT LOCATION: (IN 12 LV SEGMENTS)

EACH \odot = 1% OF LV INFARCTED.
8 \odot 's IN A SEGMENT = 100% OF SEGMENT

TOTAL POINTS 32 Each Point = 31 LV INFARCTED

If 1 criteria in bracket met, select 1 with most points
Criteria in bracket with same score are "ored" i.e. even
if 2 or more criteria met, the point is scored only once.

Figure 3.1

Selvester's 54 criteria/32 point scoring system for infarct sizing. The point scores obtained from the patient are utilized to calculate the total loss of the myocardial muscle as every point represents 3% loss of the left ventricular muscle.

The latest version of the 54 criteria/32 points scoring system including recent modifications (Selvester 1989), was used for calculation of the ECG predicted EF as follows;

$$\text{ECG Predicted EF} = 65\% - (3 \times \text{QRS score})$$

3.2.2 Old myocardial infarction

Consecutive patients with a history of myocardial infarction (MI) more than three months old were included in the study provided that they had post-infarct angina which did not respond to medical treatment thereby necessitating further clinical investigation. Reversible myocardial ischemia was demonstrated in all the patients by (ECG) treadmill and/or thallium exercise tests. The exclusion criteria in this study population included left bundle branch block, left ventricular hypertrophy, valvular heart disease and atrial fibrillation. Computerized measurements of the amplitude and the duration of the ECG waves were used for QRS scoring and calculation of the PTFV1. However, the amplitude and duration of notched waves were measured manually.

Percutaneous left heart catheterization and coronary angiography were performed as described previously (Grossman and Barry 1988). Contrast left ventriculography (LV gram) was performed using a single plane right anterior oblique view (RAO), or as biplane with left lateral and anteroposterior views. Area length measurements of RAO and biplane views were utilized to calculate the end-systolic and end-diastolic

volumes of the left ventricle (Dodge et al 1960). As premature ventricular beats interfere with the measurements of the ventricular volumes, the first normal contraction after a ventricular ectopic beat was excluded from area-length measurements. The longitudinal axis and the surface area of the end-systolic and end-diastolic frames were measured from the cineangiograms by a digitizer (Cherry CPA3 - BlBB) with reference to the external diameter of the catheter. The short axis was calculated from the following formula:

$$D = \frac{4 A}{3.14 L}$$

where D is the short axis

A is the surface area

L is the long axis

The volumes of the end-systolic and end-diastolic frames were respectively calculated from the RAO and biplane LV grams from the following equations (Rackley 1976):

$$V (RAO) = \frac{3.14 L \cdot D^2}{6} \text{ cm}^3$$

$$V (\text{biplane}) = (4/3) (3.14) (L/2) (D_{lat}/2) (D_{PA}/2) \text{ cm}^3$$

where V is the volume

L is the long axis

D is the short axis of the RAO

D_{lat} is the short axis of the lateral view

and D_{PA} is the short axis of the anteroposterior view

The ejection fractions were calculated from the following formula:

$$\text{Ejection fraction} = \frac{\text{end-diast. vol.} - \text{end-syst. vol.}}{\text{end-diastolic volume}}$$

The calculation of the PTFV1 and the Selvester QRS scores for the patients with old MI were made as mentioned above for those with acute MI.

3.3 RESULTS

3.3.1 Clinical data

Thirty-eight patients with acute MI (24 men and 14 women, mean age 55.4 and age range 36-74 years) completed the study. They included 17 patients with acute anterior and 21 patients with acute inferior MI. Their radio-nuclide EF ranged from 9% to 59% with a mean of 26.1% while the range of their QRS score was 0 - 20 with a mean of 8.23 points. Their ECGs showed the following additional features - one left axis deviation and 2 right bundle branch blocks. The relationships of the PTFV1 and QRS point scoring system to the Tc 99 EFs are discussed separately.

Thirty-three patients with old MI completed the study (23 men, 10 women, age range of 30-69 years and mean age of 53.9 years). They included 22 and 11 patients respectively with old inferior and old anterior MI. Their mean LV gram EF was 48.69% with a range of 18% - 75% while the range of their QRS scores was 0 - 18 points with a mean of 5.96 points.

3.3.2 P terminal force in lead V1 and Tc 99 EFs in acute MI

A scattergram of PTFV1 versus Tc 99 EFs in the patients with either acute anterior or inferior MI is shown in figure 3.2. The mean PTFV1 of the 38 patients was 5.2 mV.msec with a range of 0.0 to 15.1 mV.msec. The mean PTFV1 was 6.7 ± 0.42 mV.msec (range 0.0 - 15.1) in acute anterior MI and 4.0 ± 2.95 mV.msec (range 0.00 - 11.3) in acute inferior MI respectively (table 3.1). Thus the PTFV1 was significantly higher ($p < 0.05$) in patients with acute anterior compared with acute inferior MI. Moreover, the correlation of PTFV1 and Tc 99 EFs was poor in acute inferior MI ($r = 0.103$) but weakly positive ($r = 0.525$) in acute anterior MI (table 3.1).

An abnormal PTFV1 ≥ 4.0 mV.msec was found in 13/17 (76%) and 12/21 (59%) of acute anterior and acute inferior MI respectively (table 3.2). All 38 patients had Tc 99 EF $< 40\%$ except one with acute inferior MI. A PTFV1 ≥ 8.0 mV.msec was found in 8 (7 anterior and 1 inferior) out of 38 (21%) patients and all had Tc 99 EF $< 25\%$ (table 3.2). No patient in this study had clinical or electrocardiographic LVH.

3.3.3 Selvester's QRS scoring system and EF in patients with acute MI

The QRS point scores, Tc 99 EFs, predicted LV gram EFs and ECG predicted EFs of the individual patients with either acute anterior or inferior MI, are shown in tables 3.3 and 3.4. The mean QRS score as well as EFs obtained

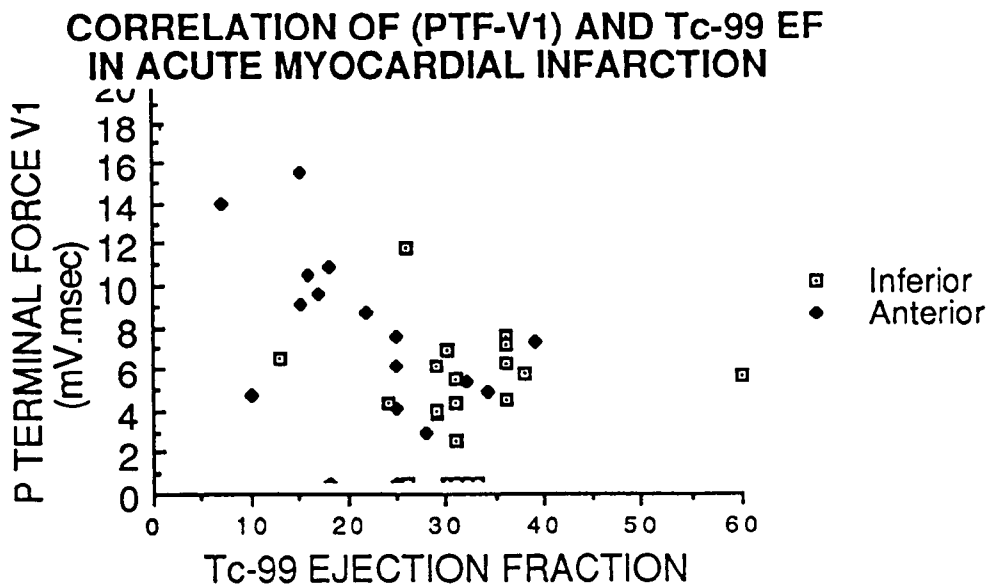


Figure 3.2

Correlation of the P terminal force in lead V1 (PTFV1) and the Technetium-99 (Tc-99) ejection fraction (EF) in patients with acute myocardial infarction (acute MI). The diamonds represent the patients with acute anterior MI ($r = -0.525$) while the squares represent the acute inferior MI ($r = -0.103$). The PTFV1 is measured as millivolt. milliseconds.

Table 3.1 PTFV1 and Tc 99 EFs of patients with acute myocardial infarction.

Infarct Site	PTFV1 (mV.msec)	Tc 99 EF (%)

Anterior		
mean	6.7 ± 0.42	20.82 ± 8.53
range	(0.00 - 15.1)	(06 - 38)
Inferior		
mean	4.0 ± 2.95	30.38 ± 8.73
range	(0.00 - 11.3)	(12 59)

Table 3.2 Technetium-99 EF and PTF-Vl in relation to the sites of acute myocardial infarction: comparison with severity of LV dysfunction.

Infarct site	TC-99 EF < 40%	TC-99 EF < 25%	PTFVl ≥ 4.0 mV.msec	PTFVl ≥ 8.0 mV.msec
Anterior n=17	17/17 (100%)	13/17 (76%)	13/17 (76%)	7/17 (41%)
Inferior n=21	20/21 (99%)	2/21 (9%)	12/21 (59%)	1/21 (4.5%)

Table 3.3 Tc 99 EFs, Predicted LV gram EFs, QRS point score and ECG predicted EFs of patients with acute anterior MI.

Patient	Sex	age(yrs)	Tc 99 EF(%)	Predicted LV gram EF(%)	QRS point score	ECG predicted EF (%)
1.	M	69	9	16.8	20	5
2.	F	49	24	34.8	10	35
3.	M	53	15	24	12	29
4.	M	57	24	34.8	8	41
5.	M	49	21	31.2	18	11
6.	F	56	24	34.8	6	47
7.	M	51	27	38.4	2	59
8.	M	58	16	25.2	5	50
9.	M	71	14	22.8	8	41
10.	F	52	17	26.4	2	59
11.	F	57	6	13.2	13	26
12.	M	40	14	22.8	8	41
13.	M	63	17	26.4	12	29
14.	M	48	24	34.8	7	44
15.	F	57	38	51.6	10	35
16.	M	46	31	43.2	8	41
17.	M	51	33	45.6	8	41

Table 3.4 Tc 99 EFs, predicted LV gram EFs, QRS point score and ECG predicted EFs in patients with acute inferior MI.

Patient	Sex	Age (yrs)	Tc 99 EF(%)	Predicted LV gram EF(%)	QRS point score	ECG predicted EF (%)
1.	M	48	23	33.6	9	38
2.	M	61	30	42	6	47
3.	F	60	29	40.8	2	57
4.	M	51	12	20.7	7	44
5.	M	64	30	42	8	41
6.	F	65	25	36	11	32
7.	M	47	25	36	15	20
8.	F	62	30	42	5	50
9.	F	74	29	40.8	12	29
10.	F	67	28	39.8	5	50
11.	F	48	28	39.8	00	65
12.	F	63	20	30	11	32
13.	M	56	35	48	8	41
14.	M	32	35	48	5	50
15.	M	65	37	50.4	1	62
16.	M	35	35	48	8	41
17.	F	47	30	42	3	56
18.	M	45	32	44.4	6	47
19.	M	55	31	43.2	7	44
20.	M	47	35	48	5	50
21.	F	54	59	76.8	12	29

from radionuclide studies, the predicted LV gram and the ECG, are shown for acute anterior and inferior MI (table 3.5). The QRS score correlated poorly with Tc-99 in acute anterior MI ($r = 0.338$) and not at all in acute inferior MI ($r = -0.007$) which resulted in a wide discrepancy between the Tc-99 ejection fractions and the QRS scores in both the anterior and inferior groups (figures 3.3 and 3.4). The correlation of the QRS score with predicted LV gram EFs was also poor in acute anterior and non-existent in acute inferior MI (r values of -0.335 and -0.007 respectively). Statistically significant differences ($p < 0.05$) were observed between Tc-99 and ECG predicted EFs both in acute anterior and inferior MI. There was no significant difference between ECG and LV gram predicted EFs in acute inferior MI although there was a significant difference in the anterior group. However, despite the absence of significant differences between the predicted LV gram and ECG predicted EFs in the patients with acute inferior MI, the confidence intervals were wide (-8.57 to 5.46) which indicates that large differences could occur in individual patients with respect to predicted LV gram and ECG predicted EFs. A QRS score ≥ 2 points was found in 16 (94%) out of the 17 acute anterior and 18 (85%) of the 21 acute inferior MI patients all of whom had a Tc 99 EF $\leq 35\%$. No patient with acute anterior MI had a score < 2 although 10% of the patients with acute inferior MI had a score < 2 but an EF $\leq 35\%$ (table 3.6).

Table 3.5 Mean + SD and range of QRS score EFs (Tc-99, Predicted LV gram, ECG-predicted) of the patients with acute MI.

Infarct Site	Tc 99 EF (%)	Pred. LV gram EF (%)	QRS score points	ECG EF (%)

Anterior				
mean	20.82 + 8.53	30.98 + 10.24	9.23 + 4.8	37.29 + 14.4
range	(6-38)	(13.2-51.6)	(2-20)	(5-59)
Inferior				
mean	30.38 + 8.73	42.47 + 10.4	6.95 + 3.85	44.85 + 11.11
range	(12-59)	(20.4-76.8)	(0-10)	(20-65)

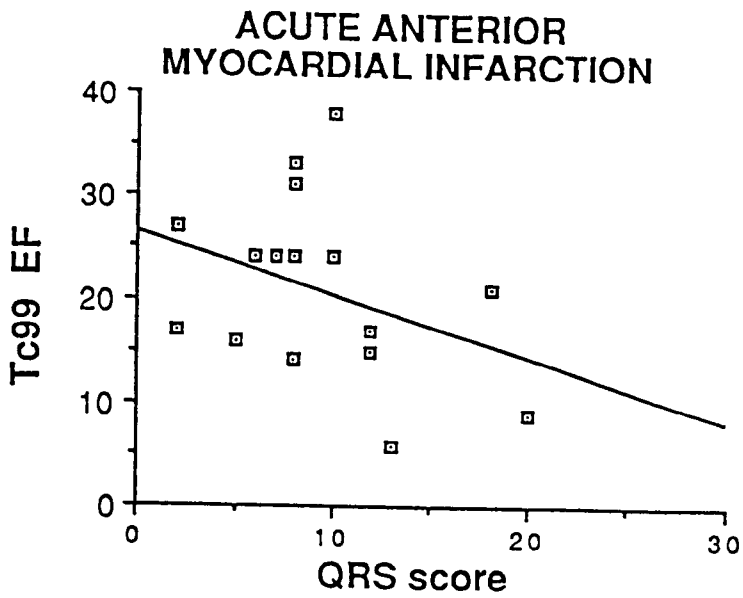


Figure 3.3

Correlation of Selvester's QRS score with Technetium-99 Ejection Fraction (Tc-99 EF) in patients with acute anterior myocardial infarction. There was a poor correlation ($r = -0.338$).

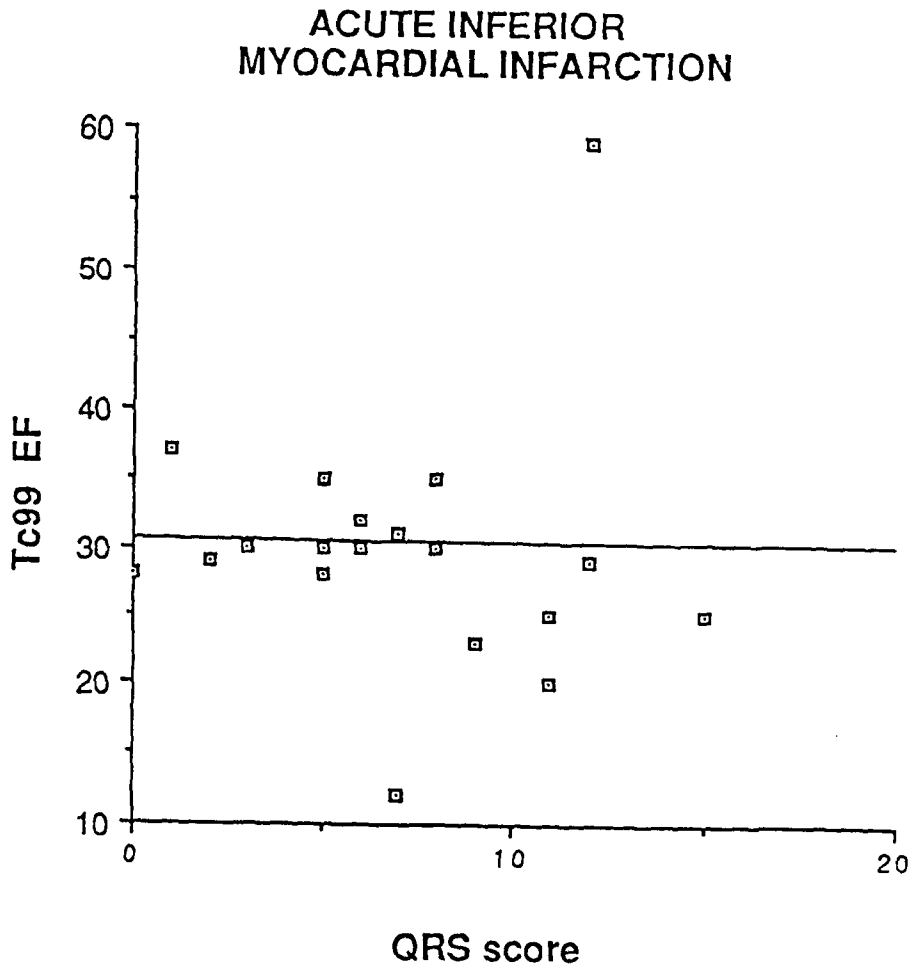


Figure 3.4

Correlation of Selvester's QRS score with Technetium-99 Ejection Fractions (Tc-99 EF) in patients with acute inferior myocardial infarction. There was no correlation ($r = -0.007$).

Table 3.6 QRS score ≥ 2 in LV dysfunction in acute
 myocardial infarction.

QRS Score	Infarct Size	Tc 99 EF \leq 35%	Tc 99 EF $>$ 35%

> 2	Anterior	16 (94%)	1 (6%)
	Inferior	18 (85%)	nil
< 2	Anterior	nil	nil
	Inferior	2 (10%)	1 (5%)

3.3.4 Catheterization and angiographic data in patients with old MI

3.3.4.1 Old inferior MI

The extent of significant coronary artery disease (narrowing $\geq 70\%$), left ventricular end-diastolic pressure (LVEDP) and ejection fraction (EF) were assessed in all patients. All the patients had triple vessel disease (3VD) except two, one of whom had disease in the right coronary artery (RCA) and the left anterior descending (LAD) while the other had a diseased RCA. The mean LVEDP in the old inferior MI patients ranged from 2 - 30 mm Hg with a mean of 11.13 mm Hg, while their EFs ranged from 18% to 75% with a mean of 47.7% (table 3.7). Only two patients in the inferior MI group had an EF < 35%, their values being 18% and 28% respectively.

3.3.4.2 Old anterior MI

There were 5 patients who had triple vessel disease (3VD), one patient with diseased RCA and LAD (2VD), and 5 patients with single vessel disease (1VD) affecting the LAD only. The mean LVEDP was 8.9 mm Hg with a range of 3 - 15 mm Hg, while EFs ranged from 25% to 75% with a mean of 51.2% (table 3.8). The LVEDP was significantly higher in patients with old inferior MI ($p < 0.05$) than in those with old anterior MI. However, there were no significant differences between the two groups of patients with old MI with regard to contrast LV gram EF using a non-paired t test.

Table 3.7 Catheterization, angiographic and electrocardiographic data of the patients with old inferior MI.

No.	Age (yrs)	Sex	Catheterization Data			Electrocardiographic Data		
			AP	LVEDP (mm Hg)	CAD	EF%	QRS score	EF% PTFV1 mV/msec
1.	48	M	132/80	20	3VD	36	3	56 0.34
2.	56	F	120/72	30	3VD	51	0	65 8.48
3.	56	M	160/90	10	2VD	72	6	47 5.16
4.	51	F	100/60	6	3VD	32	6	47 1.45
5.	61	M	144/80	7	3VD	60	4	53 3.36
6.	52	M	120/70	5	1VD	57	1	62 2.32
7.	41	M	180/60	22	3VD	48	0	65 2.04
8.	52	M	160/80	2	2VD	45	15	20 0.00
9.	39	M	110/80	12	3VD	52	8	41 4.8
10.	56	M	110/70	5	3VD	42	18	11 1.86
11.	58	M	165/80	12	3VD	75	4	53 0.00
12.	69	F	125/70	7	3VD	28	6	47 1.47
13.	41	M	125/75	7	3VD	61	6	47 0.00
14.	49	M	125/75	8	3VD	65	4	53 5.00
15.	61	F	180/80	15	3VD	18	7	44 3.48
16.	56	M	180/90	12	3VD	36	9	38 3.15
17.	59	F	140/76	10	3VD	37	5	50 2.00
18.	59	M	120/70	10	3VD	32	10	35 4.97
19.	54	M	160/60	10	3VD	56	6	47 3.40
20.	56	M	150/70	10	3VD	34	5	50 2.76
21.	48	F	150/85	17	3VD	43	7	44 2.44
22.	61	M	130/70	8	3VD	63	6	47 1.68

Table 3.8 Catheterization, angiographic and electrocardiographic data of the patients with old anterior MI.

No.	Age (yrs)	Sex	Catheterization Data				Electrocardiographic Data			
			BP	LVEDP	CAD	EF%	QRS score	EF%	PTFV1	
1.	58	M	110/70	4	3VD	25	10	35	4.76	
2.	42	M	124/80	15	1VD	39	5	5	3.06	
3.	53	M	116/72	15	1VD	31	12	29	3.06	
4.	53	F	140/72	10	1VD	41	6	47	5.18	
5.	54	F	150/80	5	1VD	49	6	47	5.40	
6.	52	M	180/95	10	3VD	75	3	50	3.70	
7.	58	M	160/80	3	3VD	66	2	59	4.10	
8.	59	M	120/80	7	3VD	67	4	53	1.9	
9.	50	M	130/80	12	1VD	66	7	44	1.36	
10.	68	F	140/60	9	3VD	61	3	56	1.86	
11.	56	M	140/75	8	2VD	44	3	56	3.76	

3.3.5 P terminal force in lead V1 in patients with old MI

In patients with old inferior MI, the PTFV1 ranged from 0.00 to 8.48 mV.msec with a mean of 2.72 mV.msec, while in the old anterior MI group it ranged from 1.36 to 5.40 mV.msec with a mean of 3.28 mV.msec (table 3.9). The PTFV1 in the patients with old anterior MI was significantly higher ($p < 0.05$) than in the patients with old inferior MI. However, the PTFV1 correlated well with the LVEDP ($r = 0.449$) in patients with old inferior MI, but it did not correlate with the LVEDP ($r = 0.525$) in patients with old anterior MI in view of the smaller number of patients. Moreover, the PTFV1 did not correlate with the contrast LV gram ejection fraction in both old anterior ($r = 0.383$) and old inferior MI ($r = 0.061$).

3.3.6 Selvester's QRS score in patients with old MI

The QRS score ranged from 2 to 10 with a mean of 5.54 in the patients with old anterior MI, while the patients with old inferior MI had a mean QRS score of 6.2 with a range of 0 to 18 (table 3.9). There were no significant differences between the two groups of patients with old MI with respect to the QRS score (non-paired t test). However, the QRS score correlated well but negatively with the contrast LV gram ejection fractions in the patients with old anterior MI ($r = - 0.707$) but there was a poor correlation ($r = 0.252$) between the QRS score and the contrast LV gram ejection fractions in the patients

Table 3.9 Mean, standard deviation and range of LVEDP, contrast LV gram EF, QRS score, ECG predicted EF and PTFV1 of old anterior and old inferior MI patients.

Site of MI	LVEDP (mm Hg)	LV gram EF (%)	QRS score	ECG predicted EF (%)	PTFV1 (mV.msec)
Anterior mean \pm SD	8.9 \pm 4.06	51.2 \pm 16.5	5.54 \pm 3.1	47.8 \pm 9.1	3.46 \pm 1.36
n=11	3-15	25-75	2-12	29-59	1.9-5.4
Inferior mean \pm SD	11.1 \pm 6.42	47.4 \pm 15.08	6.18 \pm 4.22	44.9 \pm 14.4	2.73 \pm 2.06
n=22	2-30	18-75	0-18	11-65	0-8.48

with old inferior MI. Moreover, there was a good correlation ($r = 0.637$) between the ECG predicted EFs and the contrast LV gram EFs in patients with old anterior MI but not in the patients with old inferior MI ($r = 0.242$).

3.4 DISCUSSION

3.4.1 P terminal force in lead V1 and LV dysfunction in MI

Morris and associates (1964) studied 100 normal subjects and 87 patients with aortic and mitral valve diseases. They found that the product of the amplitude and the duration of the negative deflection of the P wave in lead V1 which they called "P terminal force in lead V1" was more negative than $- 0.03 \text{ mm} \cdot \text{sec}$ ($= - 3 \text{ mV} \cdot \text{msec}$) in only 2.5% of the normal population but in 92% of patients with left sided valvular heart disease. Romhilt and Scott (1972) noted that the signs of left atrial abnormality satisfying the Morris criterion (1964) for an abnormal PTFV1 were present in 76% of patients during acute episodes of pulmonary oedema, of which half were reversible after a few days. These changes may indicate that volume and pressure overload rather than the atrial mass is responsible for changes in PTFV1 (Chung 1980a). An abnormal PTFV1 $\geq 0.03 \text{ mm} \cdot \text{sec}$ has been found to correlate highly with raised pulmonary capillary wedge pressure in patients with acute MI (Heikkila et al 1973, Chandraranta and Hodge 1978). Although the preload and left ventricular filling pressures are important factors

affecting left ventricular function, the ejection fraction represents a global function of the left ventricle which can be evaluated non-invasively to categorize patients with acute MI into those with good and bad prognosis. However, the value of the PTFV1 in the assessment of LV function has not been studied in patients with acute MI.

In this study, there was a negative correlation between the PTFV1 and Tc 99 EF in patients with acute anterior MI ($r = -0.525$) but the PTFV1 did not correlate with Tc 99 EF in patients with acute inferior MI ($r = 0.103$). The PTFV1 was significantly higher ($p < 0.05$) in patients with acute anterior compared to those with acute inferior MI. All patients had a Tc 99 EF $< 40\%$ except one with acute inferior MI. The results in table 3.2 reveal that the magnitude of the PTFV1 appeared to reflect the severity of LV dysfunction in acute MI, particularly in the anterior group. The latter was shown by the finding that severe LV dysfunction i.e. EF $< 25\%$ was associated with a criterion of PTFV1 ≥ 8 m.sec in patients with acute MI (3rd day postinfarct).

The mean LVEDP was significantly higher ($p < 0.05$) in patients with old inferior MI than in those with old anterior MI (table 3.9). Furthermore the PTFV1 correlated well with the LVEDP in old inferior MI ($r = 0.449$). However the correlation of PTFV1 with LVEDP in old anterior MI was poor despite a higher (r^2 0.525) than in old inferior MI ($r = 0.449$) due to smaller

number of patients with old anterior MI (tables 3.7 and 3.8). This finding suggests that PTFV1 correlates with the LVEDP in patients with old MI only when there is associated LV dysfunction with increased preload. This also demonstrates the association of abnormal PTFV1 and raised left atrial pressure overload. The paradoxical correlation of the PTFV1 with LV dysfunction in acute and old MI with respect to the site of the MI (anterior or inferior) is probably related to the extent of the infarct. However in the acute stage of myocardial infarction, associated myocardial ischemia adds to the LV dysfunction while the ischemic injury is not represented in the QRS scoring system.

3.4.2 Selvester's QRS scoring and LV function in MI

Prognosis after MI and functional reserve capacity are related to the infarct size. Ideker et al (1978) have shown that infarct size and LV function are inversely related with a high correlation ($r = 0.88$). LV EF was also found to be one of the most important prognostic factors after MI (Hammermeister, De Roun and Dodge 1979). The adverse prognostic effects of both infarct size and poor LV function are well known. Patients with infarct sizes approaching 40% with EF < 35% have an annual mortality rate of more than 20% (Nelson et al 1975, Geltman et al 1979, CASS Principal Investigators and Associates 1983).

Left ventricular ejection fraction can be calculated from contrast LV grams (Dodge et al 1983),

echocardiograms (Pombo et al 1971), and radionuclide studies (Ashburn et al 1978). However in comparison with the previous investigations, the ECG is non-invasive, cheap and easily reproducible. Myocardial infarction causes diminution of R wave and/or appearance of the Q wave (Hillis et al 1976, Prinzmetal et al 1954, Shaw et al 1954). The QRS point scoring system has been designed to predict infarct size and LV function in patients with old MI (Selvester 1989). The Selvester QRS scoring system was found to correlate well with infarct size in old anterior (Ideker et al 1982), old inferior (Roark et al 1983) and old posterolateral infarcts (Ward et al 1984).

An extended 54 criteria/32 points system was found to correlate better with infarct size than the original 37 criteria/29 points system (Selvester et al 1982a, Selvester et al 1982b). Selvester (1989) also suggested an equation from which EF can be predicted from the QRS point score. However, it remained unclear whether the QRS scoring system could predict with a reasonable degree of accuracy the EF in individual patients with acute MI especially so because the 54 criteria/32 points system has not been widely studied particularly with respect to the site of the infarct. In addition other studies have shown that the 37 criteria/29 points scoring system correlates well with radionuclide EF on the third day (Palmeri et al 1982) and > 3 months post infarct (DePace et al 1982). An increase of the QRS score on day 1, 2

and 3 after acute inferior MI is expected (Anderson et al 1983). Therefore, for the present study, ECGs recorded on the 3rd day post-infarct were used.

Development of the QRS system and its validation was based on contrast biplane ventriculograms (Hindman et al 1985, Selvester et al 1978). As radionuclide scans underestimate EFs, the ECG predicted EFs were also compared with predicted LV gram EFs from the regression equations used in our laboratory and poor correlation was obtained. The reasons for the differences in Tc 99 and predicted ECG EFs (table 3.5) are not clearly understood but probably are due to the fact that the QRS scoring system represents infarct size (necrotic tissue) while the EF is a function of the viable myocardium. The results in tables 3.3 - 3.6 reveal that the 54 criteria/32 points scoring system is of limited value in the prediction of EF in individual patients with acute MI. However the system was useful in separating patients with acute MI and good LV function (score < 2 points) from those with LV dysfunction (score \geq 2 points). Loss of viable myocardium leads to suppression of LV function as was demonstrated in this study by finding that a QRS score \geq 2 points was associated with Tc 99 EF < 35% in 94% and 85% of acute anterior and inferior MI patients respectively.

A low score with significant LV dysfunction observed in two patients might have been due to additional ischemic left ventricular dysfunction as ECG signs of

ischemia are not represented in the QRS score. The equation for calculation of ECG predicted EF uses the mean of EFs (65%) for all patients which might explain the variations in ECG predicted EFs; the mean contrast LV gram EF equals 0.67 ± 0.08 (SD) in normal subjects (Rackley et al 1980). Furthermore, EF is affected by many other factors not reflected by the QRS score such as the loading conditions of the LV (preload and afterload), circulatory catecholamines which are raised in acute MI, heart rate and medications such as betablockers, calcium channel blockers, nitrates and anti-arrhythmic drugs.

The reason for the poor correlation of QRS score and contrast LV gram EFs in old inferior MI is not known but probably one explanation is QRS underscoring, because it is well established that electrocardiographic changes of inferior MI may regress or even disappear with time due to overgrowth of viable myocardium on the myocardial scar.

6/33 (18%) patients with old MI had contrast LV gram EFs < 35% of the order of 28%, 18%, 32%, 34%, 25%, and 31% with QRS scores of 4, 7, 10, 5, 10 and 12 respectively (tables 3.7 and 3.8). Therefore it was not possible to utilize the QRS scores to separate patients with old MI into those with good LV function and LV dysfunction because there were many other patients who had higher QRS scores with good EFs.

3.5 CONCLUSIONS

It can be concluded that:

(I) The magnitude of the PTFV1 appears to reflect the severity of LV dysfunction in patients with acute MI. A value of ≥ 8 mV.msec was found in 8/38 (21%) of the patients with acute MI (7 anterior and 1 inferior) and all had Tc 99 EF $< 25\%$.

(II) The PTFV1 in old inferior MI associated with extensive coronary artery disease, correlated well with the LVEDP. This finding as well as (I) may have implications for the ECG diagnosis of left ventricular hypertrophy by affecting the point scoring system of Romhilt-Estes.

(III) The 54 criteria/32 point scoring system (3rd day post-infarct) is helpful in separating patients with acute MI with good LV function (score < 2 points) from those with LV dysfunction (score ≥ 2 points).

(IV) Utilization of the QRS scoring system did not allow classification of patients with old MI into those with good LV function and LV dysfunction.

(V) At this stage it is difficult to use the ECG scoring system to predict accurately the EF from the ECG in individual patients either with acute or old myocardial

infarction.

In summary, Selvester's 54 criteria/32 points scoring system cannot be utilized to predict EF from ECG in individual patients with acute or old MI. Nevertheless, this scoring system was useful in separating patients with acute MI who had good LV function from those with LV dysfunction. However, Selvester's 54 criteria/32 points scoring system was not able to classify patients with old MI into those with good LV function and others with LV dysfunction. This is the first study which has showed that severe LV dysfunction i.e. $EF < 25\%$ can be predicted from the ECG with a criterion of $PTFV1 \geq 8$ mV.msec in patients with acute MI (3rd day post-infarct).

4. ISOLATED REPOLARIZATION WAVE ABNORMALITIES AND SYMPTOMATIC CORONARY ARTERY DISEASE

4.1 INTRODUCTION

Repolarization abnormalities are recognized features of coronary artery disease (CAD) particularly when associated with pathological Q waves and/or ST segment deviations. However, T wave abnormalities without pathological Q waves and/or ST segment deviations are not uncommon and their interpretation in relation to CAD is open to question, especially in the inferior leads, particularly aVF. A wide angle between the QRS and T wave axis has been claimed to separate normal and abnormal T wave changes in the frontal plane. In particular, Schamroth (1976) suggested that a QRS-T angle > 60 degrees as an isolated criterion is an indication of myocardial disease, including ischemia. This is supported by Chung (1980b), but Rowlands (1980) suggests that an angle > 45 degrees between the QRS and T wave axes is abnormal (figure 4.1). Therefore, it was decided to study the role of QRS-T angle in a group of patients with documented symptomatic CAD with particular emphasis on T wave abnormality in the inferior leads and to compare the findings with those derived from a group of apparently healthy individuals.

4.2 METHODS

Patients who had symptomatic CAD for which they were to undergo coronary arteriography were considered for

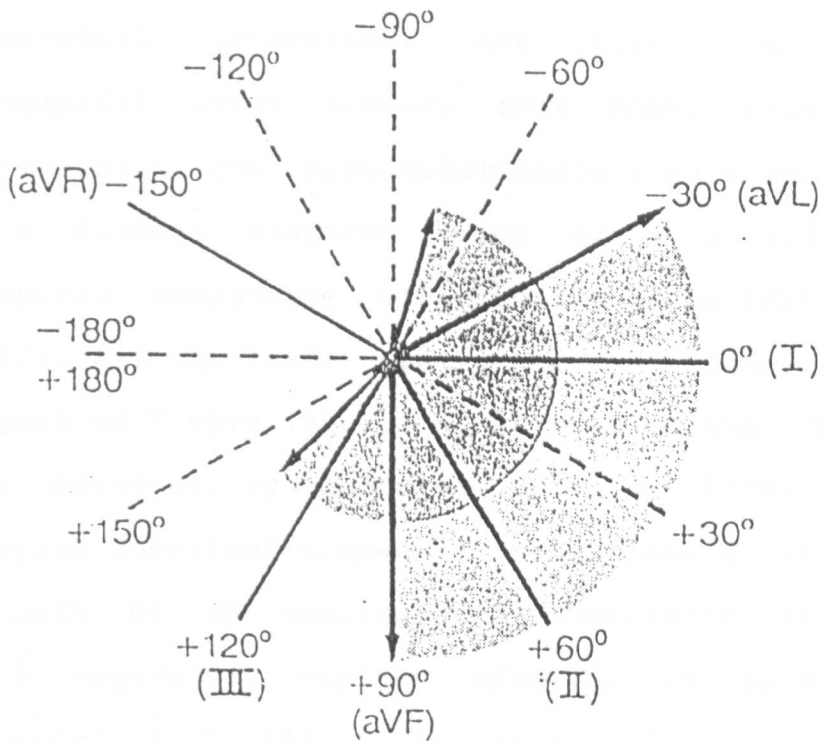


Figure 4.1

Rowlands criteria for normal QRS-T angle. In this figure it is shown that the normal range of the QRS axes is from -30 to $+90$ degrees (light shading), while for the T wave axis, the normal ranges from -75 to $+135$ degrees. However, according to Rowlands, the T wave axis cannot be regarded as normal or abnormal without reference to the QRS axis. He suggests that the T wave axis is abnormal if it differs from the QRS axis by more than 45 degrees in either direction.

inclusion in the study provided that they had (i) isolated T wave inversion without pathological Q wave and/or ST segment deviations in lead aVF with or without T wave inversion in lead I, (ii) no history of old myocardial infarction, and (iii) no acquired or congenital heart disease that might cause T wave inversion. The electrocardiograms were recorded either by a Siemens Mingorec 4 or by a locally developed computer compatible electrocardiograph (Watts and Shoat 1987). All the leads were recorded simultaneously and the degree of T wave inversion, as well as the QRS-T angle, was measured by computer using a locally developed program described elsewhere (Macfarlane et al 1986). As a part of an ongoing study (Macfarlane et al 1985) of 1315 apparently healthy normals, 12 lead ECGs were recorded from 395 individuals aged ≥ 40 years (242 men and 153 women). All the healthy volunteers were screened by a physician.

4.3 RESULTS

12 patients (9 male, 3 female, age range 49-66 with a mean age of 58.7 years) with symptomatic coronary artery disease and isolated T wave abnormalities in the limb leads but no pathological Q waves and/or ST segment deviations were studied. Their QRS and T wave axes, and the QRS-T angle are shown in table 4.1.

All had significant coronary artery disease on coronary angiography (narrowing $\geq 70\%$) which revealed that 10 had triple vessel disease. The remaining two had

Table 4.1 QRS axis, T wave axis and QRS-T angle of the patients studied.

Patient	Sex	Age (yrs)	QRS axis	T-wave axis	QRS-T	T avF amplitude microvolt
1.	M	62	- 06	- 09	03	- 53
2.	M	55	06	- 12	18	- 78
3.	M	63	00	- 21	21	- 60
4.	M	61	11	- 20	31	- 113
5.	M	60	18	- 20	38	- 91
6.	M	62	11	- 39	50	- 148
7.	F	66	- 02	- 59	57	- 65
8.	M	59	46	- 15	61	- 85
9.	F	49	41	- 23	64	- 61
10.	M	60	18	- 55	73	- 88
11.	M	51	- 13	- 89	76	- 119
12.	F	56	44	174	- 130	- 51
Mean		58.7	+14.5	-15.6	30.2	- 84.4
Range		(49->66)	(13 -> +46)	(-89 -> +174)	(-130 -> +76)	(-5 -> -148)

double vessel disease; one of them had disease of the left anterior descending and circumflex arteries while the other had right coronary and circumflex arteries affected.

The results of the QRS-T angle are shown below:

1. The mean QRS axis from all patients was + 14.5 with a range of -13 to + 46 degrees while their mean T wave axis was -15.6 with a range of -89 to +174 degrees.
2. A narrow QRS-T angle below 60 degrees was found in 7/12 (58%) patients. They included 6 men and 1 woman with a mean age of 61.2 years and a range of 55-66 years. Their mean QRS axis was +5.4 with a range of -6 to +18 degrees. The mean T wave axis in these patients was -25.7 degrees with a range of -9 to -59 degrees.
3. A QRS-T angle exceeding 60 degrees (figure 4.2) was found in 5/12 (42%) patients (table 4.1) who included 3 men and 2 women with an age range of 49-60 years and a mean age of 55.2 years. The QRS axis of four of them ranged from -13 to +46 degrees with a mean of +23 degrees and their T wave axis ranged from -15 to -89 degrees with a mean of -45.5 degrees. One patient had a QRS axis of 44 degrees and a T wave axis of +174 degrees on account of T wave abnormalities in lead I.
4. A QRS-T angle less than 45 degrees (figure 4.3) was found in 5/12 (42%) patients. Their QRS axes

Table 4.2 The mean \pm SD and the ranges of the QRS-T angle in a sample of 1315 apparently healthy subjects.

Age Group (years)	Sex	Number of individuals	QRS-T angle (degrees)
18-29	Male	265	16.6 \pm 23.9 (-39 - 71)
	Female	317	12.5 \pm 25.1 (-49 - 59)
30-39	Male	220	7.2 \pm 27.3 (-61 - 59)
	Female	118	10.6 \pm 21.7 (-47 - 61)
40-49	Male	117	-4.8 \pm 26.3 (-67 - 37)
	Female	73	-13.0 \pm 27.1 (-82 - 40)
50 +	Male	125	-13.0 \pm 27.8 (-82 - 40)
	Female	80	-15.0 \pm 27.8 (-89 - 26)

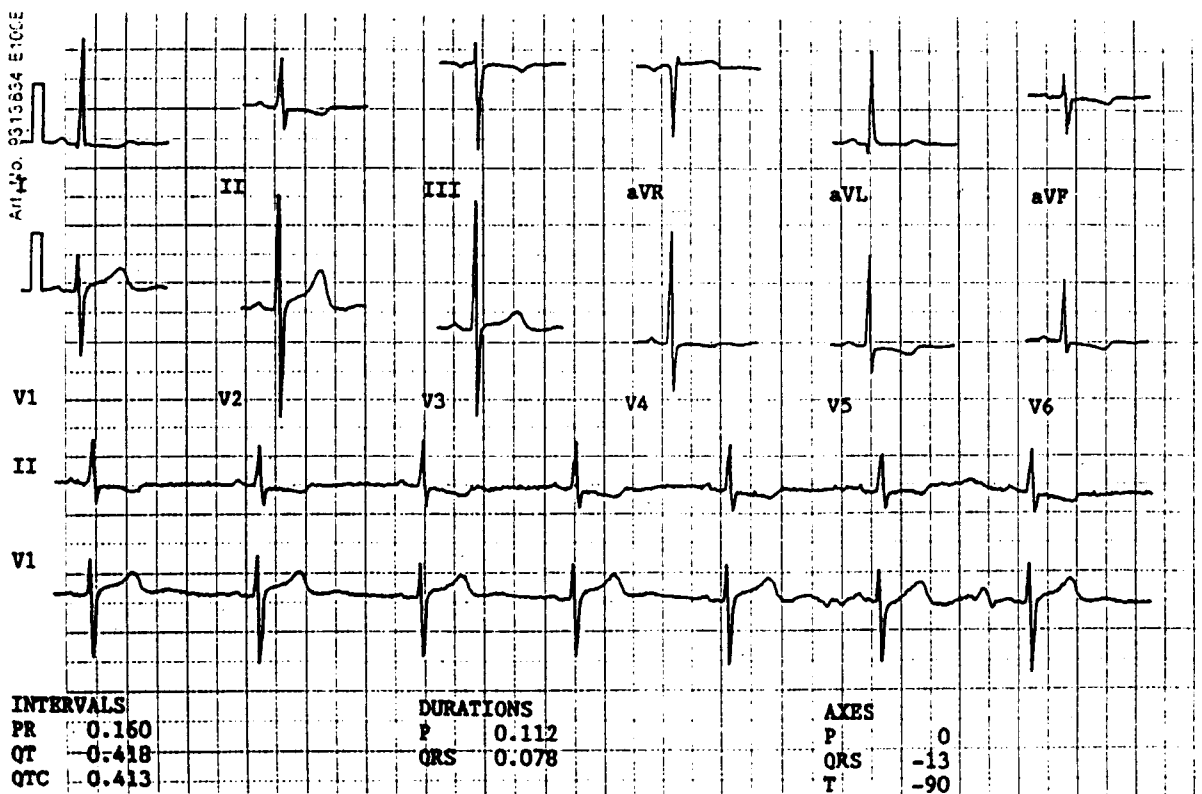


Figure 4.2

Wide QRS-T angle and symptomatic coronary artery disease. Electrocardiogram of a 53 year old male showing T wave inversion in leads I, II, III, aVF, V5 and V6 due to triple vessel disease documented by coronary angiography. The QRS-T angle was 77 degrees.

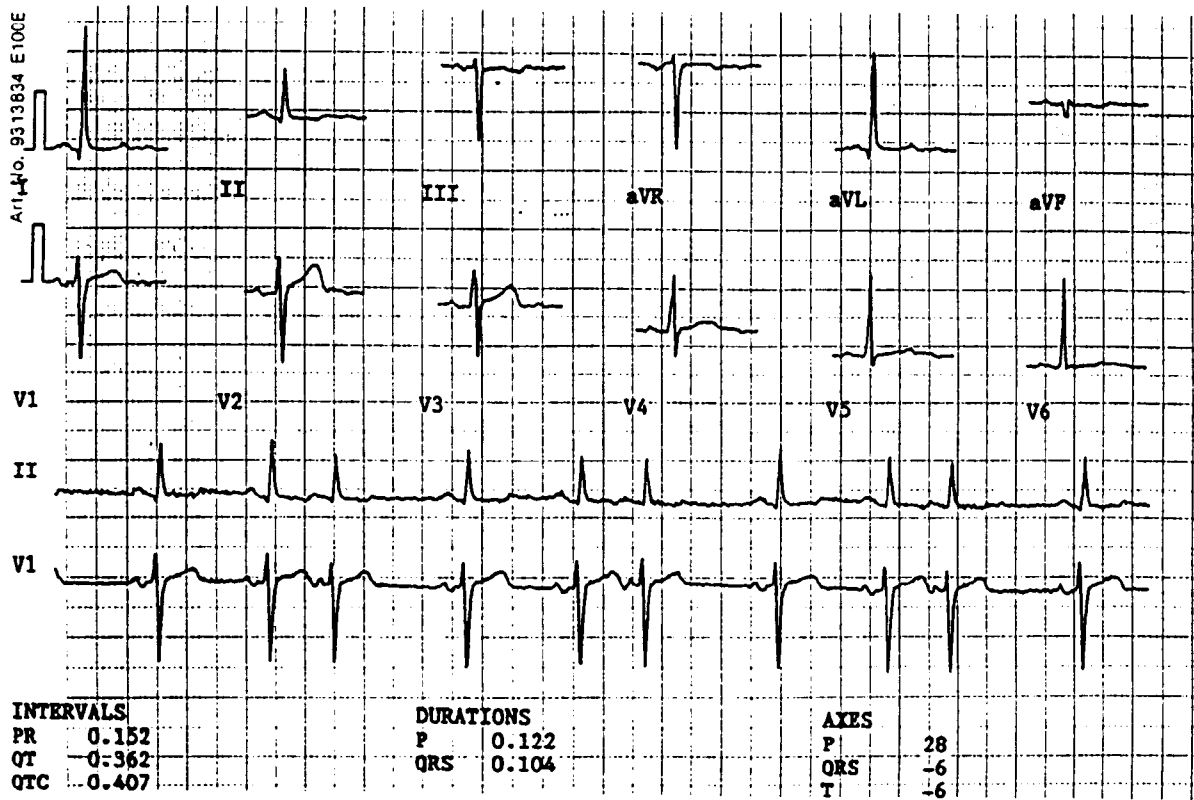


Figure 4.3`

Narrow QRS-T angle and symptomatic coronary artery disease. Electrocardiogram of a 62 year old male patient showing T wave inversion in leads II, III and aVF due to triple vessel disease documented by coronary angiography. The QRS-T angle was zero degree (< 45 degrees).

ranged from -6 to +18 with a mean of +5.8 degrees while the T wave axis ranged from -9 to -20 degrees with a mean of -16.4 degrees.

5. A QRS-T angle > 45 degrees was found in 7 patients. The QRS axis in 3 of them ranged from -13 to +46 with a mean of +16.8 degrees while their T wave axis ranged from -15 to -89 with a mean of -46.6 degrees.
6. The normal range of QRS-T (table 4.2) and the criterion of QRS-T > 60 degrees was also assessed in a population of 1315 apparently normal individuals.
7. All patients had T wave inversion in lead aVF with a range of -50 to -148 microvolts and a mean of -84.4 microvolts. Eleven had a T wave axis < 0 degrees and hence the combination of QRS-T > 60 degrees and T < 0 was assessed as was the pairing of QRS-T > 45 and T < 0 degrees. Results are shown in table 4.3.
8. The results in table 4.3 indicate that the use of QRS-T angle $> 45^\circ$ or 60° as an isolated criterion for myocardial ischemia has low predictive value of 15-20% only. While T wave axis 0° is shown to be highly specific (98%) and sensitive (92%) with a predictive value of 64% for myocardial ischemia addition of the criterion QRS-T axis to T axis $< 0^\circ$ lowered the predictive value. When T axis $< -15^\circ$ was used to diagnose CAD, it was 100%

Table 4.3 Evaluation of QRS-T angle with or without T wave axis in a population ≥ 40 years. Criterion were assessed in two different populations: (i) 12 patients with coronary artery disease ≥ 40 years and (ii) 397 apparently healthy individuals ≥ 40 years.

Criterion	False positives in the normals	Specificity	Sensitivity	Predictive Value
QRS-T $> 60^\circ$	20/397 (5%)	377/397 (95%)	5/12 (42%)	5/25 (20%)
QRS-T $> 45^\circ$	41/397 (10%)	356/397 (90%)	7/12 (58%)	7/48 (15%)
T axis $< 0^\circ$	6/397 (1.5%)	391/397 (98%)	11/12 (92%)	11/17 (64%)
QRS-T $> 60^\circ$ and T $< 0^\circ$	5/397 (1.2%)	392/397 (99%)	4/12 (33%)	4/9 (44%)
QRS-T $> 45^\circ$ and T $< 0^\circ$	5/397 (1.2%)	392/397 (99%)	6/12 (50%)	6/11 (54%)
T axis $< -15^\circ$	0/397 (0%)	397/397 (100%)	8/12 (66%)	8/8 (100%)

specific, 66% sensitive and thus had a predictive value of 100%.

9. The criterion $T < 0^\circ$ in lead aVF was further evaluated in a population of 920 apparently healthy individuals < 40 years. There were 14/920 (1.5%) of those subjects who had $T < 0^\circ$ in lead aVF. However, QRS-T $> 60^\circ$ was observed in 8/920 (0.8%) of this population thereby providing a specificity of 99.3% for myocardial ischemia in this age group. Therefore, the specificity of the isolated criterion $T < 0^\circ$ in lead aVF as an index of myocardial disease improved from 98.4% to 99.3% when combined with QRS-T $> 60^\circ$. Furthermore, no subject in this population had a T wave axis $< -15^\circ$. Therefore, a new criterion of $T < -15^\circ$ is proposed for the detection myocardial ischemia in a population < 40 years of age with 100% specificity (table 4.4).

4.4 DISCUSSION

T wave inversion simulating myocardial ischemia may occur in healthy individuals as a normal variant such as the persistence of the juvenile pattern in V2, V3 which is more commonly seen in black women (Thomas et al 1960) and the benign T wave inversion of healthy young adults especially in young black and trained athletes (Gottlieb et al 1975, Hane-Paparo et al 1971, Lichtman et al 1973, Chou 1986b). However, in all the above reported types of T wave inversion found in young people, the T wave inversion was mainly in the precordial leads and

Table 4.4 Evaluation of the criteria $T < 0$ mV in lead aVF, $QRS-T > 60^\circ$ and T axis $< -15^\circ$ in 920 apparently healthy individuals < 40 years of age.

Criterion	No. of individuals with positive criteria	Specificity

$T < 0$ mV	14/920 (1.5%)	98.4%
$QRS-T > 60^\circ$	8/920 (0.8%)	99.3%
T axis $< -15^\circ$	0/920 (0%)	100%

occasionally associated with changes in inferior leads. It is also well known that premenopausal women may exhibit non-specific T wave changes particularly in the inferior leads.

Myocardial ischemia may be defined electrocardiographically as a condition in which (i) the depolarization process takes place normally (ii) repolarization is delayed in a particular area and (iii) polarization is normal during diastole (Lipman, Massie 1969b). The repolarization sequence of the ventricles is directed from epicardium to the endocardium while the repolarization vector points in the opposite direction to that of the repolarization sequence (contrary to that of depolarization). The QRS and T vectors are therefore both directed from the endocardium to the epicardium, i.e. in the same direction in a normal heart. Therefore the direction of the QRS and T axes are normally similar so that the QRS-T angle < 60 degrees. In the presence of myocardial disease - commonly ischemia - the T wave axis tends to deviate away from the ischemic region whereas the QRS axis remains normally directed. Therefore a QRS-T angle > 60 degrees is thought to be a sensitive index of significant T wave abnormality and myocardial disease and is more reliable than the empirical observation of T wave changes in an isolated lead (Schamroth 1976).

In this study, all patients had symptomatic ischemic heart disease proved by coronary angiography. They were

all selected on the basis of having T wave inversion in lead aVF, while one had T wave inversion in lead I in addition. These patients were compared with a population of apparently healthy individuals.

However, in the apparently healthy individuals, the use of a single criterion such as the QRS-T angle exceeding either 45 or 60 degrees have low predictive value for CAD (table 4.3). It therefore seemed sensible to improve criteria by adding the requirement that the T axis in the frontal plane should be superior to 0 degrees. When this was done as shown in table 4.3, it was found that the sensitivity decreased slightly but the specificity improved. Thus a new criterion of a T axis < 0 degrees combined with QRS-T > 45 degrees is proposed as consistent with myocardial ischemia for individuals ≥ 40 years.

Therefore the addition of the criterion $T < 0^\circ$ to the criterion QRS-T $> 45^\circ$ and QRS-T $> 60^\circ$ in age group ≥ 40 years increases the specificities of the latter two criteria from 90% and 95% respectively to 99% for both criteria (table 4.3). The combination of a wide QRS-T axis exceeding 45 degrees together with a T axis superior to 0 provided a higher sensitivity (50%) compared to the sensitivity of 33% for the criterion of QRS-T $> 60^\circ$ and $T < 0^\circ$. Therefore its application to subjects with or without symptoms of ischemic heart disease can be recommended, as it is 99% specific for myocardial ischemia for those ≥ 40 years. However the criterion T

axis $< 0^\circ$ performed much better than QRS-T angle with or without T axis less than 0° (table 4.3). The criterion T axis $< 0^\circ$ is 98% specific for coronary artery disease with a sensitivity of 92% and a predictive value of 64%. Furthermore the criterion T $< -15^\circ$ had even higher predictive value than T $< 0^\circ$ as its ability to predict CAD was 100% in a population aged 40 years and over. This new criterion i.e. T $< -15^\circ$ is to be preferred. The results in table 4.4 reveal that T < 0 mV in lead avF is not common as it was observed in only 14 (1.5%) of the 920 apparently healthy individuals. Therefore the criterion of T $< 0^\circ$ has a specificity of 99.4% for myocardial ischemia in a group of asymptomatic individuals. However no subject among the 920 apparently healthy individuals had T < 0 mV in lead avF with T wave axis $< -15^\circ$. Therefore the criterion of T < 0 in avF with T axis $< -15^\circ$ is 100% specific for myocardial disease in a population of asymptomatic individuals < 40 years. However isolated repolarization wave abnormalities are not uncommon in hospital and general practice. Therefore the diagnostic role of the criterion T < 0 mV in lead avF and T axis $< -15^\circ$ in this population of patients may vary from that of a population of asymptomatic healthy individuals < 40 years of age.

The overall conclusion would seem to be that the use of QRS-T angle as an isolated criterion has a low predictive value for both cut off points of 60° and 45° . The criterion T axis $< 0^\circ$ is introduced for the diagnosis

of ischemic heart disease with high specificity and sensitivity. However when the T wave axis criterion is modified from $T < 0^\circ$ to $T < -15^\circ$, the predictive value rises from 64% to 100% but with a consequent drop in sensitivity from 92% to 66%. Therefore the use of the criterion $T < -15^\circ$ is recommended both in patients below and above 40 years of age.

5. ECHOCARDIOGRAPHIC MEASUREMENTS ON DIFFERENT CONVENTIONS

5.1 INTRODUCTION

Accurate echocardiographic measurements of the left ventricular (LV) dimensions are essential for the calculation of left ventricular mass (LVM) and left ventricular volume (LVV) which are important parameters for the definition of left ventricular hypertrophy (LVH) and left ventricular dilatation (LVD).

The recommendation of the American Society of Echocardiographers, i.e. ASE (Sahn et al 1978) and the so called Penn Convention (Devereux and Reichek 1977) are widely used for measurement of LV dimensions. The ASE recommendations are that M-mode measurements should be made from the leading edge of one wall to the leading edge of another at the onset of the QRS complex (figure 5.1). On the other hand, according to the Penn convention, measurements should be made at the peak of the R wave with the endocardial echoes of the interventricular septum and posterior left ventricular wall being excluded. These echoes however are included in measurement of the left ventricular internal diameter (figure 5.1). Thus it is clear that in view of the different techniques in measuring left ventricular internal diameter (LVID) for example, significant differences must occur in the calculation of left ventricular volume and hence, mass.

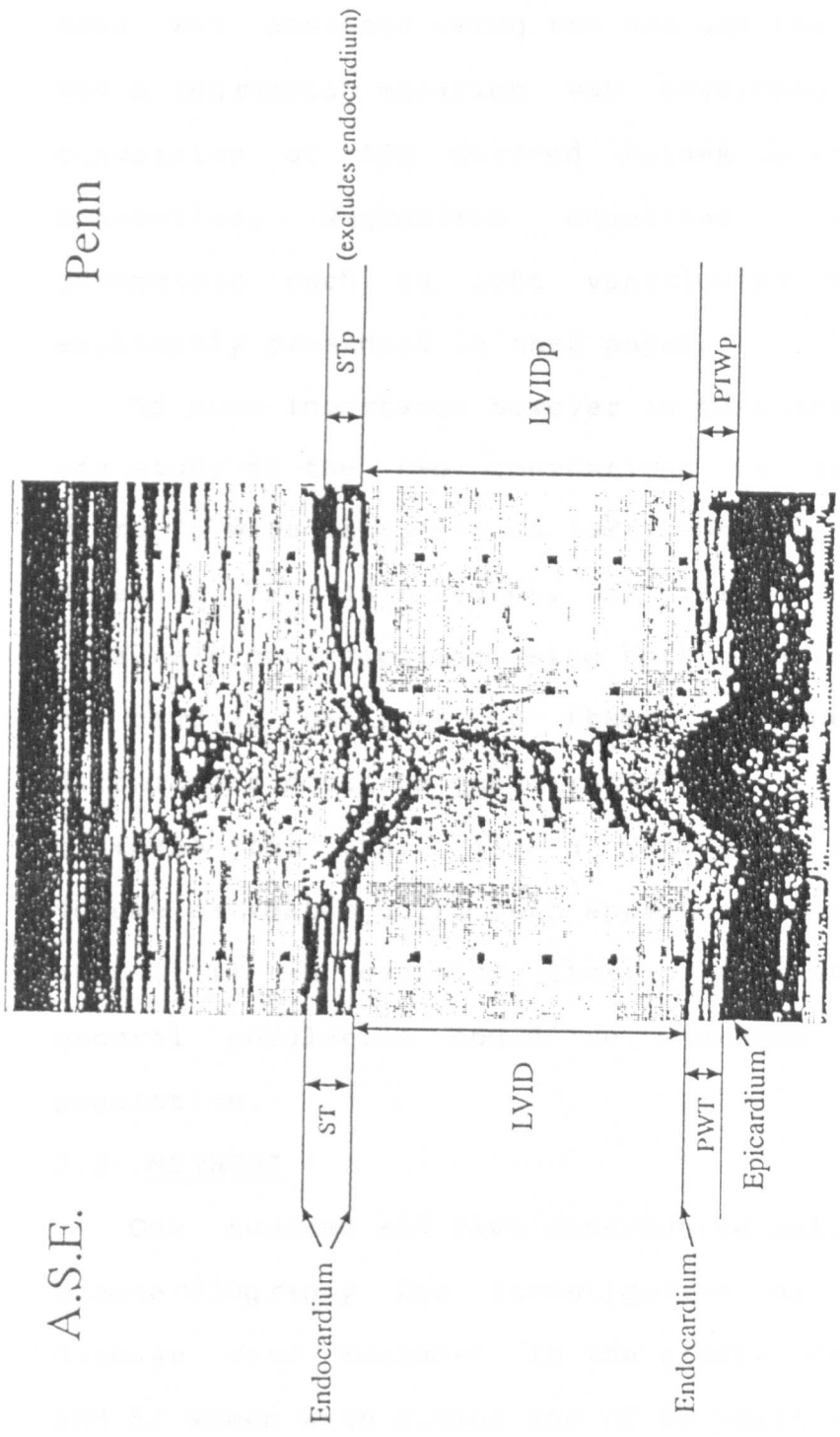


Figure 5.1 Measurement of left ventricular (LV) dimensions according to the Penn convention and the recommendations of the American Society of Echocardiographers (ASE). The details are given in the text.

Indirect comparisons of both techniques have been made in a sample of the general population in Framingham (Savage et al 1987). In particular, left ventricular mass was assessed using the ASE and the Penn convention and a regression equation was developed which allowed conversion of ASE derived values to those of the Penn convention. Regression equations relating other parameters such as left ventricular volume were not explicitly presented in that paper.

Of more importance however is that there has not been any study of the two conventions in cardiac patients where a wide variation in left ventricular parameters is expected. For this reason, it was felt essential to measure echocardiograms using both techniques in a sample of cardiac patients so that; (i) the extent of the differences could be quantitated prior to developing ECG criteria for LVH, (ii) a regression equation linking volume estimates using both approaches could be derived, and (iii) an equation linking estimates of mass in a general population could be assessed in a cardiac population.

5.2 METHODS

One hundred and five consecutive patients undergoing echocardiography for investigation of cardiovascular disease were included in the study. There were 53 men and 52 women with a mean age of 52 years (range 16 - 80 years). The distribution of the underlying pathological conditions is listed in table 5.1.

Table 5.1 Distribution of the underlying pathological
 conditions in the study population.

Cardiovascular Disorder	Number of Patients

Valvular heart disease	49
Hypertension	19
Coronary artery disease	11
Congestive cardiac failure	10
Miscellaneous	<u>16</u>
Total	105

The echocardiograms were recorded with a Diasonics Cardiovue 3400R phased array imaging system using a 2.25 MHz transducer. The patients were positioned semisupine in the 45 degree left lateral position and the echo windows that best demonstrated the septum and posterior wall were chosen in the 3rd, 4th or 5th intercostal space in the left parasternal area. M-mode recordings were obtained and measurements of the interventricular septum (IVS), posterior wall of the left ventricle (PWL) and left ventricular internal dimension (LVID) were made at end diastole according to the recommendations of ASE and Penn conventions. Measurements were made to the nearest millimetre.

Left ventricular volume (LVV) was calculated from the following equation (Macfarlane 1986):

$$LVV = \frac{3.14 \times (LVID)^3}{3} \quad (cm^3)$$

This equation was used both with the ASE recommendations and the Penn convention in order to compare volumes calculated according to the different methods of measuring LVID.

The left ventricular mass (LVM) was calculated from the equations of Troy et al (1972) and the Penn convention (Devereux and Reichek 1977) as follows:

$$LVM (ASE) = 1.05 [(IVS+PWL+LVID)^3 - (LVID)^3] \text{ g}$$

$$LVM(Penn) = 1.04 [(IVS+PWL+LVID)^3 - (LVID)^3] - 13.6 \text{ g}$$

where LVID etc. have different values according to the measurement convention used.

The volumes and masses were then indexed to body surface area (BSA) which was obtained from the following equation (DuBois and DuBois 1916):

$$BSA = 0.0001 (71.84) \times (Wt^{0.425}) \times (Ht^{0.725})$$

where BSA is in meter², weight (Wt) in Kg and height (Ht) in centimetres. For example a subject with left ventricular mass of 171g and a body surface area of 1.8 m² has an indexed left ventricular mass of 95 g/m². In order to compare the left ventricular mass obtained directly from the Penn convention with that derived indirectly from regression equations based on mass derived from ASE recommendations, the following equations from the Framingham study (Savage et al 1987) were used:

$$Mass(Penn) = 0.93 \times Mass(ASE) - 17.92 \text{ g/m}^2 \text{ (for males)}$$

$$Mass(Penn) = 0.88 \times Mass(ASE) - 9 \text{ g/m}^2 \text{ (for females)}$$

All the ASE and Penn derived LV dimensions, indexed LVV and LVM were compared by using a paired t test as were the Penn convention masses derived directly and indirectly.

A regression equation was developed for conversion of LVV calculated from ASE based measurements of the LVID to Penn equivalents and vice versa.

5.3 RESULTS

The mean and standard deviations and ranges of the various measurements derived according to both conventions are shown in table 5.2.

ASE measurements of interventricular septum, posterior left ventricular wall thickness and indexed left ventricular mass were significantly higher than those of the Penn convention while the left ventricular internal diameter and indexed left ventricular volume were significantly lower ($p < 0.001$) than the corresponding Penn data. The mean differences of the corresponding measurements are also shown in table 5.3 and 5.4.

The distribution of the differences of the LV dimensions (IVS, PWLV and LVID) indexed left ventricular volume and mass as measured directly by the two methods are shown in figures 5.2, 5.3, 5.4, 5.5 and 5.6 respectively.

Left ventricular volumes estimated by the two techniques were highly correlated ($r > 0.9$) and thus a regression equation for linking the indexed volume derived from the ASE measurements to that derived from the Penn convention was also obtained as follows:

$$\text{Vol (ASE)} = 0.9325 \times \text{Vol (Penn)} - 2.4784 \text{ cm}^3$$

When the left ventricular mass estimates based on ASE Recommendations were converted indirectly to Penn

Table 5.2 Mean \pm standard deviation and the range of echocardiographic dimensions (IVS, PWLV, LVID), indexed LVV and index LVM on ASE and the Penn conventions.

Echo Convention	IVS(cm)	PWLV (cm)	LVID (cm)	LVV/BSA ml/m ²	LVM/BSA gm/m ²
ASE	1.33+0.47 0.5 - 2.7	1.17+0.38 0.5 - 2.7	5.25+1.03 2.9 - 8.1	97.38+55.98 16.8 - 325	199.04+88.10 87 - 489
Penn	1.15+0.44 0.4 - 2.5	1.07+0.3 0.4 - 2.6	5.44+1.02 3.1 - 8.4	107.0+59.6 18.5 - 362	170.9+79.4 70 - 407.7

Table 5.3 Differences between ASE and Penn convention based measurements presented as Penn - ASE. IVS = interventricular septum, PWLV = posterior wall left ventricle, and LVID = left ventricular internal dimension at end-diastole. No regression equations were used in these calculations which were made directly from the measured data.

	IVS (cm)	PWLV (cm)	LVID (cm)
mean \pm SD	-0.183 \pm 0.112	0.085 \pm 0.92	0.190 \pm 0.118
range	-0.6 to 0.0	-0.4 to 0.0	0.0 to 0.5

Table 5.4 Differences between ASE and Penn
convention based on measurements presented
as Penn - ASE, LVV/BSA = left ventricular
volume indexed to body surface area, and
LVM/BSA = left ventricular mass indexed to
body surface area. No regression equations
were used in these calculations which were
made directly from the measured data.

	LVV/BSA (ml/m ²)	LVM/BSA (g/m ²)
means \pm SD	9.708 \pm 7.458	- 28.107 \pm 17.506
range	0.2 to 37	- 21.3 to - 91.3

Distribution of IVS (Penn-ASE) Measurements

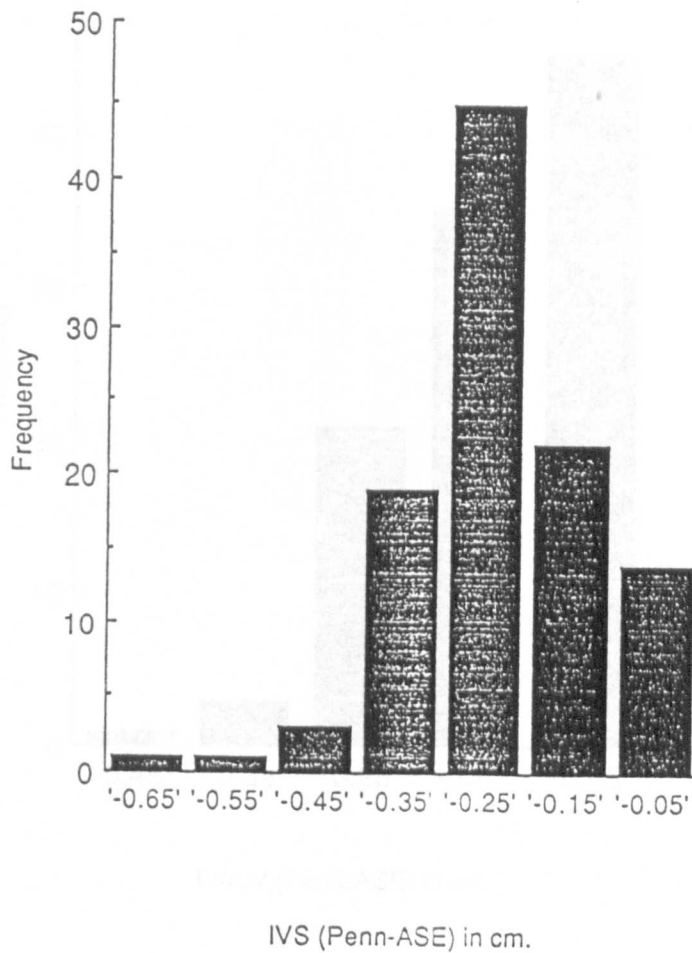


Figure 5.2

Distribution of the differences of the thickness of the interventricular septum (IVS) measured on the Penn convention and on the recommendations of the American Society of Echocardiographers (ASE). The values are represented as Penn minus ASE measurements (in centimeters).

Distribution of PWLV (Penn-ASE) Measurements

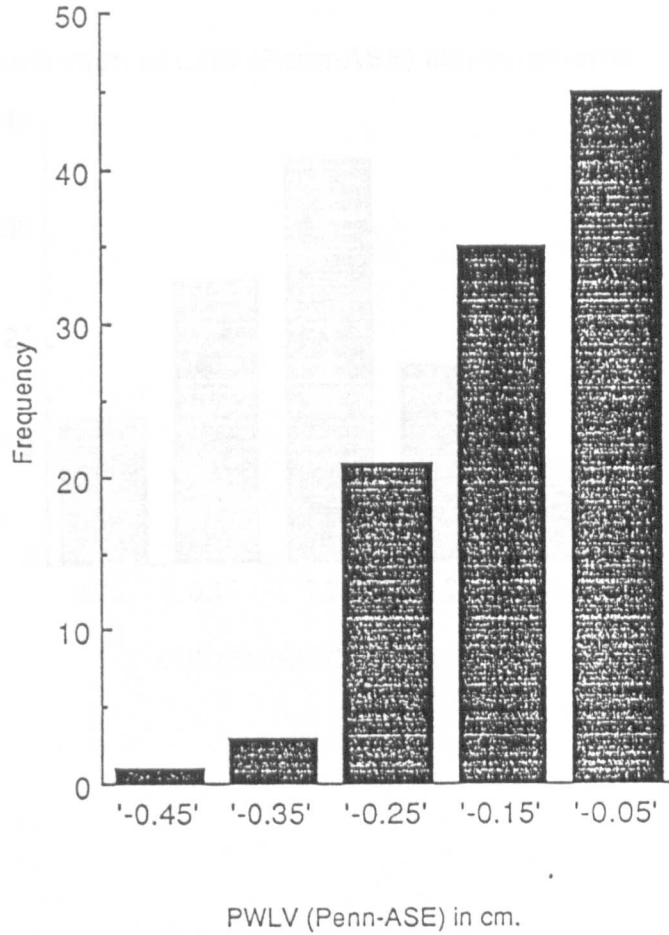


Figure 5.3.

Distribution of the differences of the thickness of the posterior wall of the left ventricle (PWLV) measured on the Penn convention and on the recommendations of the American Society of Echocardiographers (ASE). The values are represented as Penn minus ASE measurements (in centimeters).

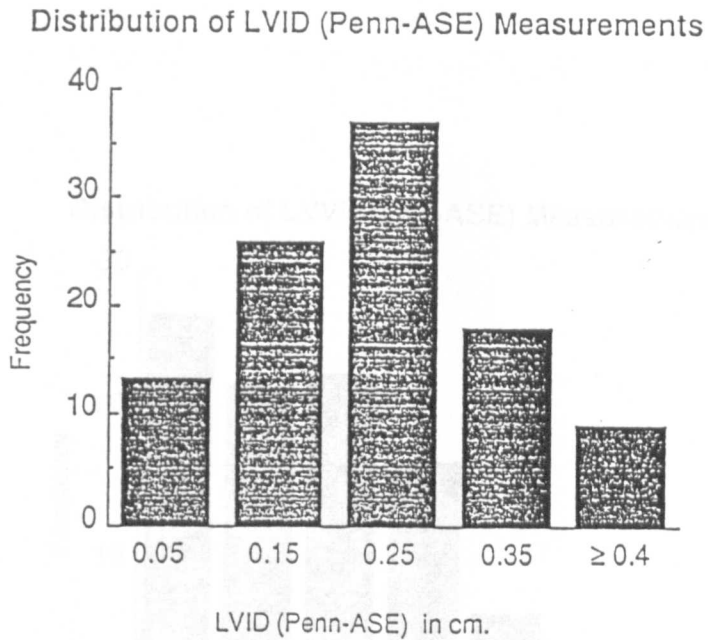


Figure 5.4

Distribution of the differences in length of the left ventricular internal dimension at end-diastole measured on the Penn convention and on the recommendations of the American Society of Echocardiographers (ASE). The values are represented as Penn minus ASE measures (in centimeters).

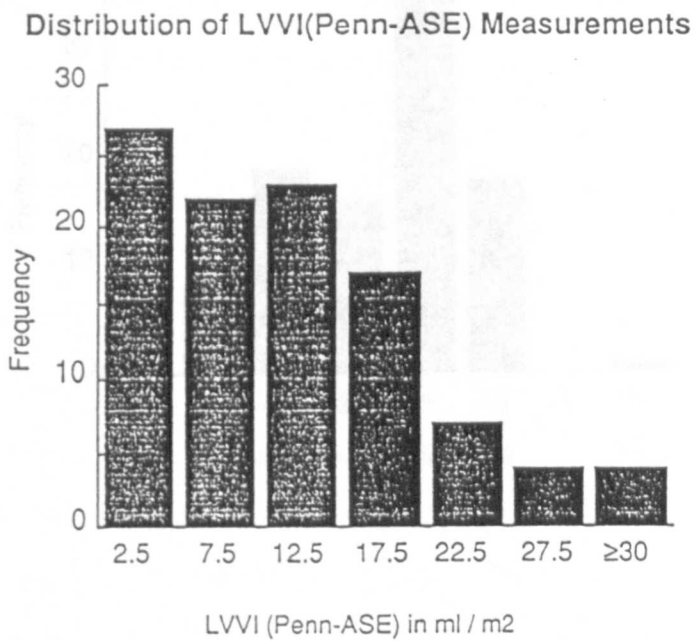


Figure 5.5

Distribution of the differences of the indexed left ventricular volume (LVVI) in ml/m^2 derived from measurements based on the Penn convention and on the ASE recommendations. The values are represented as Penn minus ASE estimates.

Distribution of LVMI (Penn-ASE) Measurements

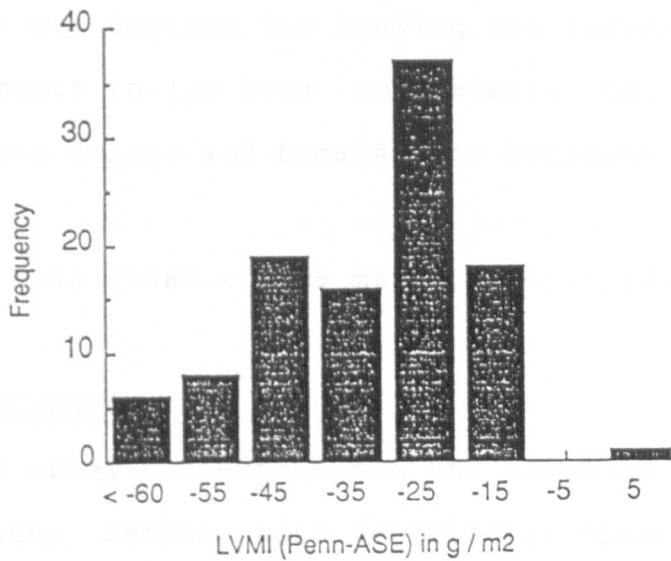


Figure 5.6

Distribution of the differences of the indexed left ventricular masses (LVMI) in g/m^2 derived from the measurements based on the Penn convention and on the ASE recommendations. The values are represented as Penn minus ASE estimates.

equivalents by the regression equation given in Methods above (Savage et al 1987) the masses tended to be significantly different ($0.05 < p < 0.1$) from those obtained directly from the Penn convention. However, there was a good correlation ($r > 0.9$) between the ASE and Penn estimates of the LVM and therefore a regression equation was derived for linking the indexed LVM from ASE measurements to its Penn equivalents for this cardiac population (males and females) as follows:

$$\text{Mass (ASE)} = 1.09 \text{ mass (Penn)} + 12.7 \text{ grams}$$

5.4 DISCUSSION

This study has emphasized the presence of significant differences between left ventricular dimensions obtained from ASE Recommendations and the Penn convention.

Echocardiography is a simple method for the calculation of left ventricular volume (Pombo et al 1971). LV dilatation has been defined as left ventricular volume $> 90 \text{ ml/m}^2$ on the basis of measurement techniques which were subsequently agreed upon by the American Society of Echocardiographers (ASE) (Pombo et al 1971).

From the regression equation derived above for linking Penn and ASE measurements of volume, the cut-off value for the upper limit of normal using the Penn convention is found to be 99.05 ml/m^2 . In general terms this would suggest that an easily remembered value of

100ml/m² should be used as the upper limit of normal left ventricular volume derived from the Penn convention.

Accurate estimation of left ventricular volume is essential for research studies because LV dilatation has prognostic significance. Furthermore LV dilatation is an adverse prognostic sign of leaking aortic and mitral valves (Henry et al 1980, Borow et al 1980). LV dilatation is also known to follow acute myocardial infarction (Hammermeister, De Roun and Dodge 1979) and both end-diastolic and end-systolic left ventricular volumes have been shown to be the most important predictors of survival after acute myocardial infarction (Hammermeister et al 1979, White et al 1980).

Hypertrophy is commonly associated with many cardiovascular diseases and constitutes a major risk factor for coronary artery disease and sudden cardiac death (Kannel and Sorlie 1981). Moreover, quantitative assessment of symmetrical left ventricular hypertrophy (LVH) in research studies is only possible with accurate estimation of the left ventricular mass (LVM). Troy et al (1972) introduced an equation for the calculation of LVM and used a convention which was later recommended by the American Society of Echocardiographers (ASE). Devereux and Reichek (1977) found that calculated LVM based on ASE recommendations overestimated left ventricular weight as measured at postmortem. As a result, these authors introduced the Penn convention in which the endocardial echoes are excluded from the

measurements of the interventricular septum and the posterior wall of the left ventricle (figure 5.1).

Left ventricular mass has also been found to be dependent on the body surface area (BSA) and the indexed LVM/BSA is significantly lower in women than in men (Devereux et al 1984). The considerable differences in LVM calculated from ASE and Penn conventions has led to the utilization of different cut-off points of LVM for the diagnosis of LVH. Levy et al (1988) defined echo-LVH as $LVM > 150 \text{ g/m}^2$ and $> 120 \text{ g/m}^2$ in men and women respectively when the LVM is calculated from the LV dimensions measured according to the ASE recommendations. On the other hand $LVM > 131 \text{ g/m}^2$ in men and $> 109 \text{ g/m}^2$ in women is regarded as echo-LVH when the LVM is calculated using the Penn convention.

Moreover, significantly higher left ventricular internal dimensions measured by the Penn convention resulted in the overestimation of volumes compared with the ASE Recommendations (figure 5.5). Thus, a new upper limit of normal volume derived from the Penn convention has had to be calculated. On the other hand, because the ASE convention overestimates the LVM compared to that measured by the Penn convention, the findings illustrate that utilization of either ASE or Penn convention has inherent limitations for simultaneous calculation of the LVV and LVM for the same subject.

In practice it is not uncommon to find both left ventricular hypertrophy and dilatation simultaneously

present and if mass is to be correlated with volume in such patients, the researcher has two options namely, (i) to adopt one convention with regression equations relating left ventricular mass or volume to the other convention as done in this study or (ii) to utilize the Penn convention for the LVM and the ASE convention for estimation of the LVV.

The original regression equation converting masses obtained by using ASE Recommendations to those in keeping with Penn standards was based on a large sample of the general population. The left ventricle masses calculated using ASE convention were converted to the Penn estimates by regression equations utilized in the Framingham study and these indirect Penn estimates were significantly higher ($0.05 < p < 0.1$) than the LVM calculated directly by the Penn convention in the cardiac population. Therefore the regression equations obtained from a sample of the general population linking LVM on ASE and Penn conventions do not readily transfer to a cardiac population and, in this study, necessitated the development of another regression equation. It is suggested that the newly derived equation presented above, should be used for a cardiac population.

The echocardiogram is very often used as a gold standard against which electrocardiographic criteria are developed. Those involved in such work should therefore be aware of the variation in estimates derived from the echocardiogram and indicate clearly in any research

publications the method by which echo measurements were obtained.

In conclusion, this particular study has highlighted the significant differences between left ventricular dimensions measured by two different techniques commonly used in clinical practice. Users of such methods should be aware of these differences when applying criteria for normality. For the purposes of this thesis, it was decided that the Penn convention would be followed in subsequent ECG-echo studies.

6. LEFT VENTRICULAR HYPERTROPHY/ENLARGEMENT: AN
ECHOCARDIOGRAPHIC CLASSIFICATION BASED ON INDEXED
LEFT VENTRICULAR VOLUMES AND MASSES

6.1. INTRODUCTION

Concentric left ventricular hypertrophy (LVH) occurs in response to pressure overload where there is a marked increase in wall thickness but no increase in left ventricular (LV) cavity size. On the other hand, in eccentric LVH, the LV dilatation due to volume overload is more significant than the increase in the LV wall thickness (Braunwald 1980, Oparil 1985, Ford 1976, De Pace 1983, Sasayama et al 1976, Ross 1974). In the Framingham study (Savage et al 1987), echocardiographically diagnosed LVH, was classified into concentric and eccentric types by utilizing a cut-off point of the relative wall thickness (RWT) so that a value $\geq 45\%$ was used to indicate concentric LVH and a value $< 45\%$ suggested eccentric LVH. Relative wall thickness is an echocardiographic index which is defined as twice the ratio of the posterior wall of the left ventricle to its internal dimension at end diastole. Eccentric LVH was further subclassified into dilated and non-dilated types according to the left ventricular internal dimension at end-diastole (indexed to sex and body surface area). Moreover, the non-dilated eccentric LVH was the most common type in that study (Savage et al 1987). This group of non-dilated eccentric LVH had

increased indexed LV masses, a relative wall thickness "less than" 45% (by definition) and normal indexed LV internal dimension at end-diastole (LVIDD) which in effect means a normal LV volume, as this is derived from the cube of the LVIDD. However, an increased LV volume is the stimulus for the development of eccentric LVH (Braunwald 1980). Therefore, there is a conflict in that the non-dilated eccentric LVH is a dubious entity. Therefore it is not clear how accurately the relative wall thickness could differentiate among the various types of left ventricular enlargement when the LV volumes and masses are used as the gold standard especially so in a sample of the cardiac population where a wide variety of left ventricular geometry is expected. Thus it was thought essential to review the definitions of echo LVH to clarify this anomaly.

6.2 PATIENTS AND METHODS

Consecutive cardiac patients undergoing echocardiography for investigation of cardiovascular disease were studied. Only patients with good echocardiographic recordings were included, and those with hypertrophic obstructive cardiomyopathy were excluded from the study.

The M-mode echocardiograms were recorded as described earlier in Chapter 5. Measurements of the interventricular septum (IVS), posterior wall of left ventricle (PWL) and left ventricular internal dimension at end-diastole (LVIDD) were made by using both the

recommendations of the American Society of the Echocardiographers (ASE) (Sahn et al 1978) and the so called Penn convention (Devereux and Reichek 1977). These conventions are illustrated in Fig. 5.1. All the measurements were made to the nearest millimeter. The following equation was used for the calculation of the relative wall thickness (Savage et al 1987).

$$\text{Relative wall thickness} = \frac{2 \times \text{PWLV}}{\text{LVIDD}} \times 100\%$$

The left ventricular volumes and masses were calculated according to ASE recommendations and the Penn Convention, and indexed to body surface area as described earlier in Chapter 5.

Left ventricular hypertrophy was diagnosed when the left ventricular mass exceeded 131 g/m^2 in men and 109 g/m^2 in women (Devereux et al 1984). Thereafter, the LVH patients were classified into concentric and eccentric types on the basis of their RWT, as previously explained

The indexed LV volumes were studied in both eccentric and concentric LVH. LV dilatation on ASE Recommendations, is diagnosed when the LV volume exceeds 90 ml/m^2 (Pombo et al 1971). There were no corresponding Penn recommendations prior to this study.

6.3 RESULTS

6.3.1 Clinical data

202 consecutive cardiac patients were included in the study. There were 116 males and 86 females with a mean age of 52.12 years (range 16-87 years). The underlying

pathological conditions are shown in table 6.1.

6.3.2 Echocardiographic measurements

The mean, standard deviation and ranges of LV dimensions on both ASE and Penn conventions are shown in table 6.2. There were statistically significant differences ($p < 0.001$) between the LV dimensions measured using the ASE and Penn conventions (paired t test). The mean, standard deviation and range of indexed LVV and LVM are shown in table 6.3. There were highly significant differences ($p < 0.001$) between LVV and LVM derived with the two different conventions (paired t test). There were 138 (68%) patients with LVH out of the 202 subjects in this cardiac population as shown in Figure 6.1. Severe LVH ($LVM > 200 \text{ g/m}^2$) was found in 52 (37%) of the 138 LVH patients.

6.3.3 Classification of LVH/enlargement by relative wall thickness

LVH patients were classified on the basis of their relative wall thickness into concentric ($RWT \geq 45\%$) and eccentric ($RWT < 45\%$) types. As a result of this classification, 56 (33%) patients had concentric and 82 (67%) had eccentric LVH. RWT and the indexed LVV correlated well ($r = -0.5778$) in the LVH population. However, 10 (18%) out of 56 patients with concentric LVH had an LV volume $> 90 \text{ ml/m}^2$ i.e. a dilated LV, while 20 (24%) of 82 patients with eccentric LVH had an LV volume $< 90 \text{ ml/m}^2$ (Figures 6.2 and 6.3). Therefore, classification of LVH on the basis of RWT in this sample

Table 3.1 Distribution of the underlying pathological states in 202 patients included in the study.

Pathological State	Number of patients

Hypertension	61
Aortic valve disease	43
Mitral valve disease	31
Mitral and aortic valve disease	16
Aortic and ischemic heart disease	08
Ischemic heart disease	05
Myocardial infarction	11
Congestive cardiac failure	11
Miscellaneous	16
Total	202

Table 6.2 The mean, standard deviation and range of the LV dimensions
 (ASE and Penn) of 202 patients in the study.

Echo Convention	IVS (cm)	PWLv (cm)	LVIDD (cm)
ASE			
mean \pm SD	1.31 \pm 0.43	1.17 \pm 0.33	5.19 \pm 1.03
range	0.50 - 2.70	0.50 - 2.70	2.90 - 8.20
	*	*	*
Penn			
mean \pm SD	1.13 \pm 0.41	1.07 \pm 0.31	5.39 \pm 1.02
range	0.40 - 2.50	0.40 - 2.60	3.10 - 8.40

* Significantly different (p < .001).

IVS: Interventricular septum

PWLv: Posterior wall of left ventricle

LVIDD: Left ventricular internal dimension at end diastole

Table 6.3 Mean, standard deviation and range of the indexed LVV and LVM of 202 patients in the study.

Echo		LVV(ASE)	LVM(Penn)
Convention		ml/m2	g/m2

ASE	mean \pm SD	93.60 \pm 55.48	193.05 \pm 81.63
	range	15.90 - 325.00	71.00 - 532.00
		**	**
Penn	mean \pm SD	104.85 \pm 60.91	165.00 \pm 75.46
	range	18.50 - 362.00	64.60 - 505.00

** Significantly different (p < 0.001)

Indexed LVV: Left ventricular volume/body surface area

Indexed LVM: Left ventricular mass/body surface area

Distribution of indexed left ventricular masses for Males and Females

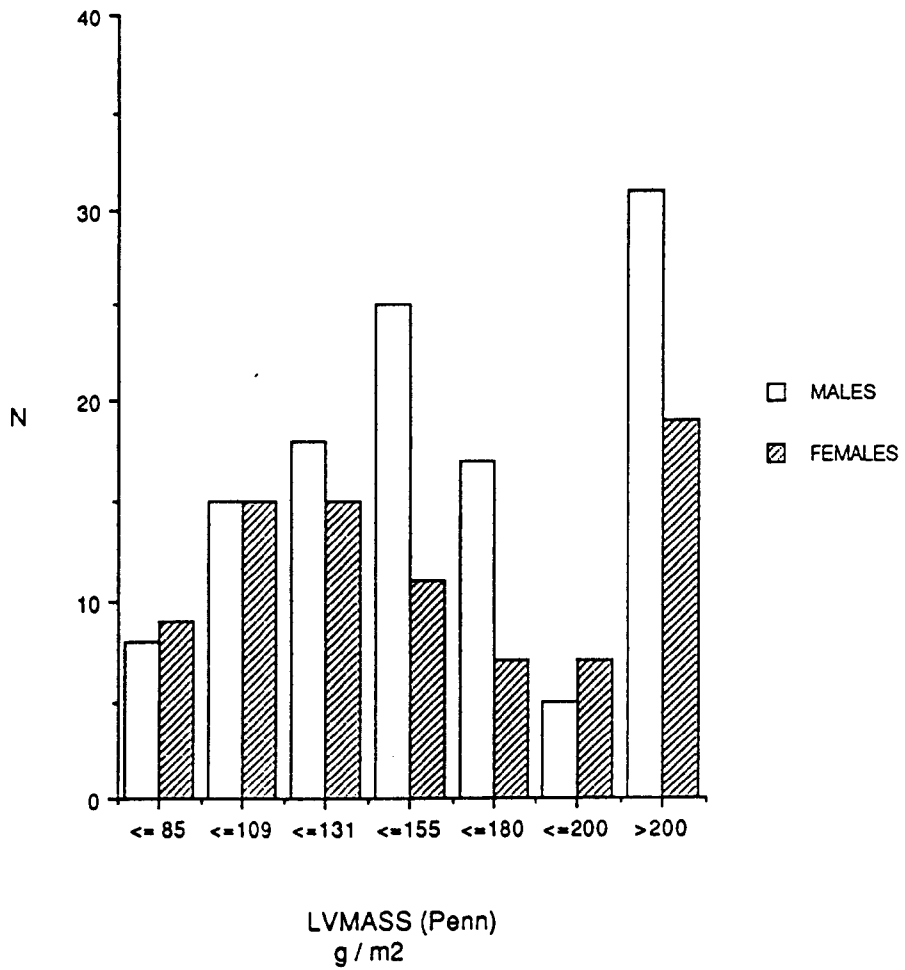


Figure 6.1

Distribution of the indexed left ventricular masses (LVMI) in g/m^2 derived from the measurements on the Penn convention for the 202 males and females included in this particular part of the study.

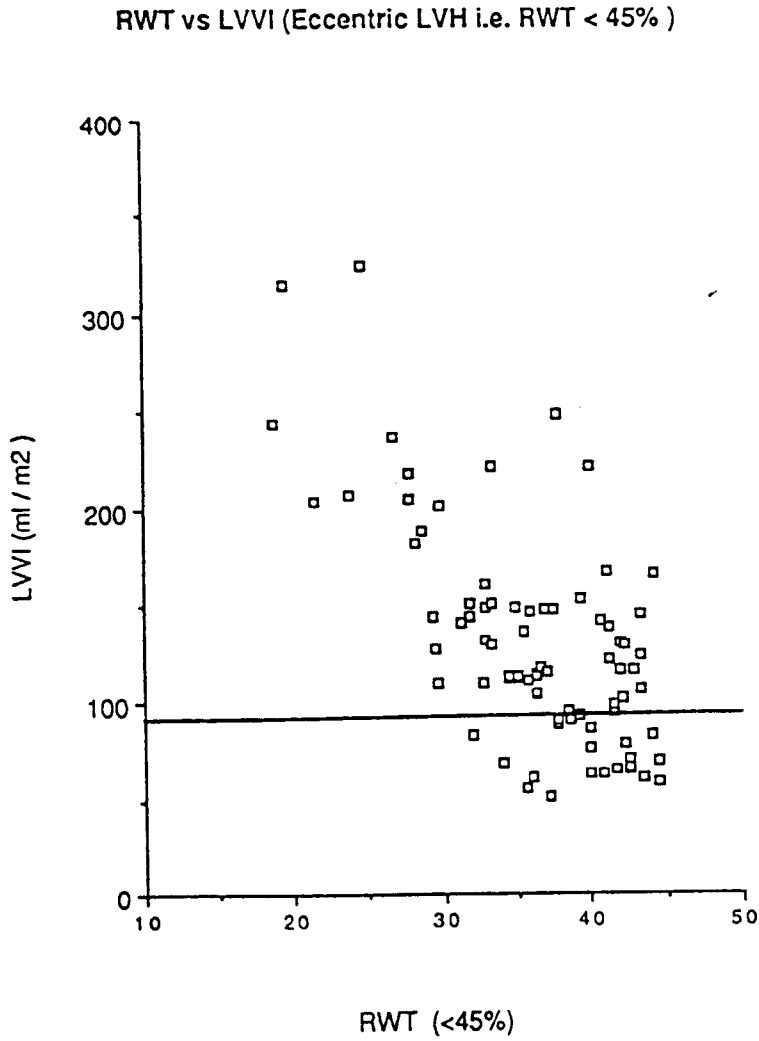


Figure 6.2

Relative wall thickness (RWT) versus indexed left ventricular volumes (ml/m²) in patients with eccentric left ventricular hypertrophy according to the Framingham classification.

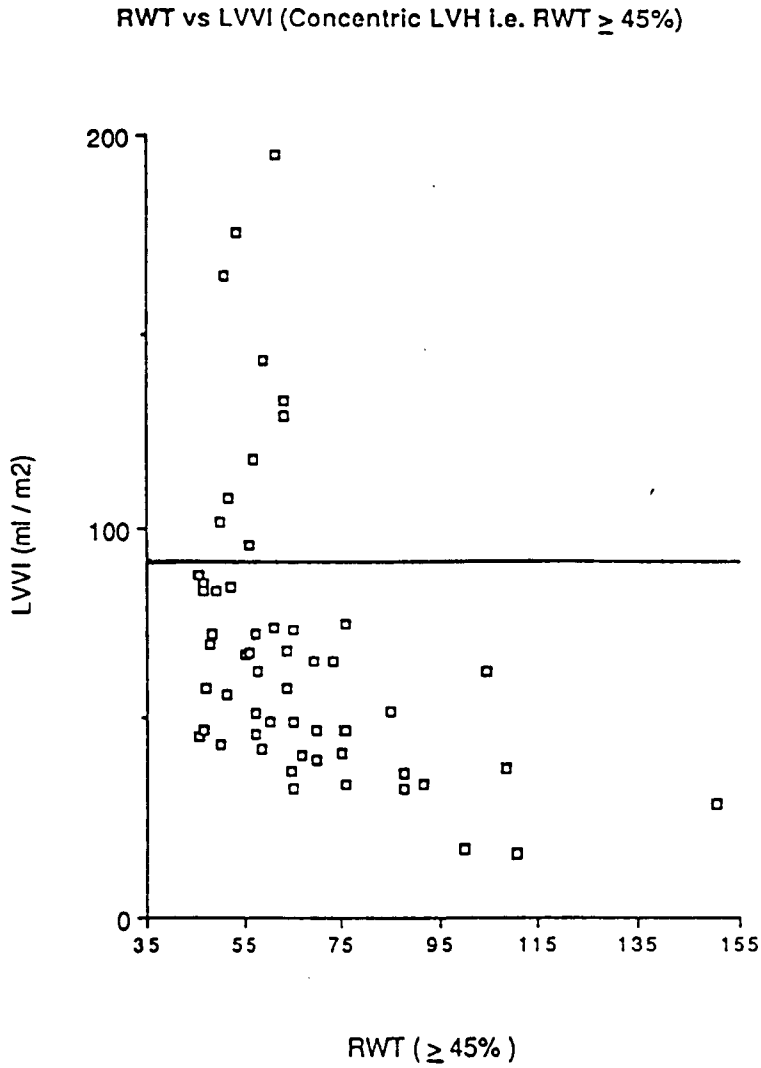


Figure 6.3

Relative wall thickness (RWT) versus indexed left ventricular volumes (ml/m^2) in patients with concentric left ventricular hypertrophy according to the Framingham classification.

of cardiac patients produced one subgroup of patients with concentric LVH and dilated left ventricles and another subgroup of patients with eccentric LVH and normal LV volume.

6.3.4 Classification of LVH/enlargement by mass/volume relationship

Because the classification of LVH on the basis of RWT produced the inconsistencies described in 6.3.3, an attempt was made to classify the patients on the basis of normal or increased left ventricular mass and volume. This allowed identification of four distinct groups (table 6.4) including a definite but small group of patients who had increased LV volume but a normal LV mass (decompensated LV). An example of an echo from this group is shown in Figure 6.4. The mean, standard deviation and range of indexed LVM (Penn) and LVV (ASE) of the four groups are shown in table 6.4. The distribution of the patients in the four groups was as follows (see table 6.4 for definitions); 54 had normal geometry, 67 had concentric LVH, 71 had eccentric LVH and 10 patients had a decompensated LV. The mean, standard deviation and range of the LV dimensions (IVS, PWLV and LVIDD) on both ASE and Penn convention of the four groups categorised on indexed LV volume and mass are shown in table 6.5.

When the RWT was studied in these four groups (table 6.6), it revealed that only 46 (68%) out of the 67 patients with concentric LVH had $RWT \geq 45\%$ while $RWT < 45\%$

Table 6.4 Mean \pm SD and range of indexed LVM and LVV (ASE and Penn) of the four echo groups obtained on the basis of mass/volume relationship

Group	Mass	Volume	LVM (Penn)	LVV (ASE)
			g/m2	ml/m2
(1) Normal	N	N	mean \pm SD	
n = 54			range	
			98.72 \pm 17.33	56.24 \pm 17.62
			64.60 - 130.00	16.8 - 88.70
(2) Concentric LVH	I	N	mean \pm SD	
n = 67			range	
			172.87 \pm 50.29	58.21 \pm 18.15
			117.00 - 349.50	15.90 - 89.00
(3) Eccentric LVH	I	I	mean \pm SD	
n = 71			range	
			219.19 \pm 81.12	148.31 \pm 49.44
			116.00 - 505.00	91.80 - 325.00
(4) Decompensated LV	N	I	mean \pm SD	
n = 10			range	
			95.53 \pm 18.00	144.14 \pm 36.70
			71.20 - 129.00	91.60 - 213.00

N:	Normal	I:	Increased
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Mass: Left ventricular mass indexed to body surface area (normally $\leq 131 \text{ g/m}^2$ in men and $\leq 109 \text{ g/m}^2$ in women on Penn Convention)

Volume: Left ventricular volume indexed to body surface area (normally ≤ 90 ml/m²) on ASE Convention).

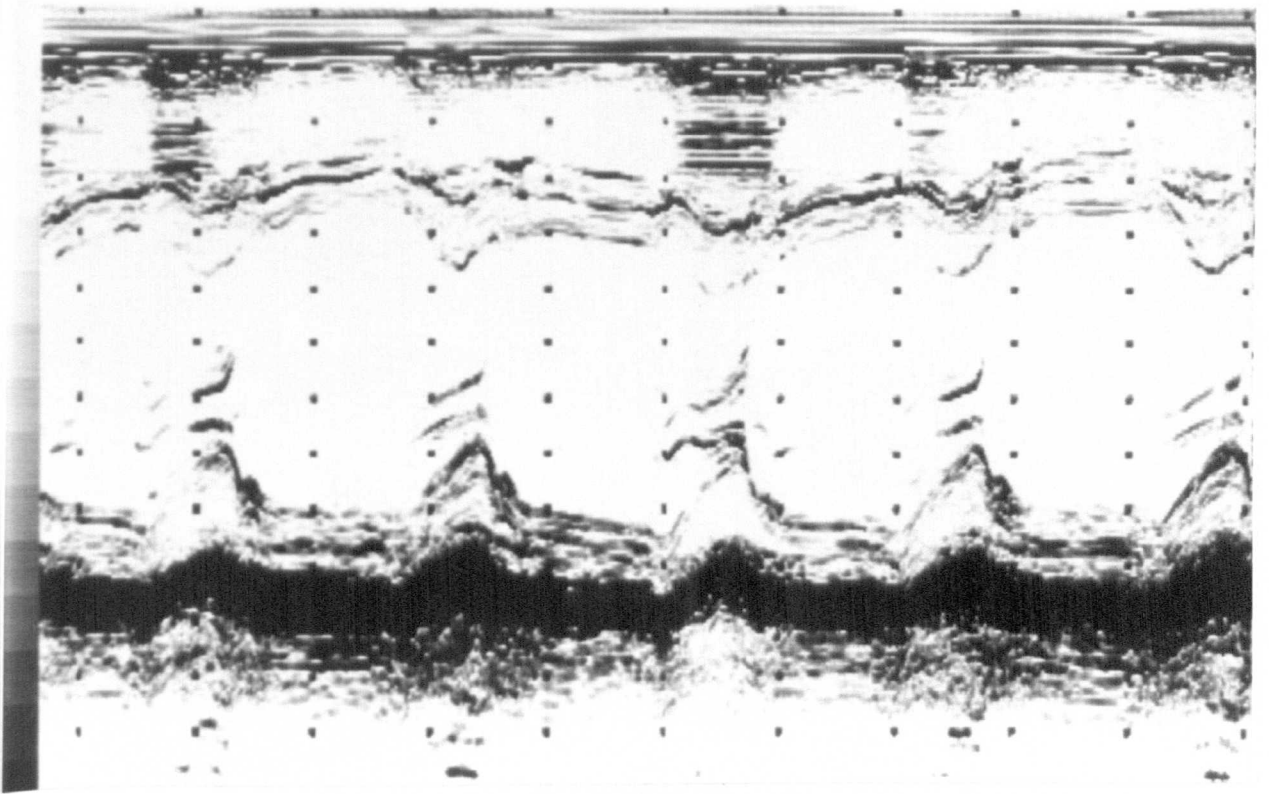


Figure 6.4

M mode echocardiogram of a patient with decompensated left ventricle diagnosed on the new classification based on mass/volume relationship. The echo shows a dilated left ventricle at the expense of thin left ventricular walls - hence a normal mass. This patient was suffering from dilated cardiomyopathy.

Table 6.5 Mean + SD and range of the LV dimensions (IVS, PWLV and LVIDD) on both ASE and Penn conventions of the four echo groups obtained on the basis of mass/volume relationship.

Group	IVS(cm)	PWLv(cm)	LVIDD(cm=
(1) ASE	1.07 + 0.23 (0.7-1.7)	1.06 + 0.23 (0.7-1.8)	4.47 + 0.5 (3.0-5.5)
Penn	0.89 + 0.22 (0.5-1.4)	0.96 + 0.21 (0.6-1.7)	4.64 + 0.50 (3.1-5.6)
(2) ASE	1.63 + 0.41 (0.9-2.6)	1.32 + 0.38 (0.8-2.7)	4.53 + 0.54 (2.9-5.5)
Penn	1.43 + 0.37 (0.7-2.2)	1.21 + 0.36 (0.7-2.6)	4.76 + 0.51 (3.3-5.7)
(3) ASE	1.28 + 0.39 (0.5-2.7)	1.16 + 0.27 (0.7-2.0)	6.22 + 0.70 (5.0-8.2)
Penn	1.11 + 0.38 (0.5-2.5)	1.08 + 0.24 (0.6-1.8)	6.41 + 0.71 (5.2-8.4)
(4) ASE	0.75 + 0.14 (0.5-1.0)	0.69 + 0.13 (0.5-1.0)	6.23 + 0.55 (5.3-7.0)
Penn	0.61 + 0.16 (0.4-1.0)	0.60 + 0.13 (0.4-0.8)	6.41 + 0.59 (5.5-7.2)

IVS: Interventricular septum (1) = Normal

PWLv: Posterior wall of left ventricle (2) = Concentric LVH

LVIDD: Left ventricular internal dimension at end diastole (3) = Eccentric LVH

(4) = Decompensated LV

Table 6.6 The relative wall thickness in the four groups of patients in this study classified on the basis of indexed LV volumes and masses.

Group			Total		RWT	
	Mass	Volume	No.	Criterion	Positive	(%)

Normal	N	N	54	$\geq 45\%$	29	52.7%
Concentric						
LVH	N	N	67	$\geq 45\%$	46	69.7%
Eccentric						
LVH	I	I	71	$< 45\%$	61	85.9%
Decompensated						
left ventricle	N	I	10	$< 45\%$	10	100%

Mass:	Left ventricular mass indexed to body surface area					
	(normal $< 131 \text{ g/m}^2$ in men and $< 109 \text{ g/m}^2$ in women)					
Volume:	Left ventricular volume indexed to body surface area					
	(normal $< 90 \text{ ml/m}^2$ on A.S.E. Convention)					
RWT:	Relative wall thickness					
N:	Normal					
I:	Increased					

was found in 61 (85.9%) of the 71 patients with eccentric LVH. Moreover, not all the patients with normal LV geometry (group 1) had $RWT \geq 45\%$. Indeed this value was found in only 26 (47.3%) of the 54 patients. The 10 patients with decompensated LV each had an $RWT < 45\%$ (mean = 22.087 ± 4.873).

6.4 DISCUSSION

6.4.1 LVH/LV dilatation versus mortality

LVH is an important risk factor for coronary artery disease (CAD) and sudden cardiac death. In the Framingham study, it has been shown that patients with electrocardiographic LVH defined as increased voltage and repolarization wave abnormalities have risks for angina and myocardial infarction (MI) similar to those patients recently recovered from an acute MI (Kannel and Sorlie 1981). Left ventricular hypertrophy may follow myocardial infarction (Rackley 1976) and LV dilatation is also a recognised prognostic factor after myocardial infarction (Hammermeister, De Roun and Dodge 1979, White et al 1987). End-diastolic volume $> 90 \text{ ml/m}^2$ was found to be more important than the ejection fraction after MI with regard to survival rate (Hammermeister, De Roun and Dodge 1979). Recently, end-systolic LVV proved to be a better indicator of survival than the end-diastolic LVV and ejection fraction after myocardial infarction (White et al 1987).

Therefore, classification of the LV geometry on the basis of LV masses and volume may reflect additive

information on the prognosis of the underlying pathological condition. There is indirect evidence to support this in patients with dilated cardiomyopathy with depressed LV function. Field et al (1973) found that both ejection fraction and mass/volume ratio (M/V) were of prognostic value and were useful indices for classification of patients with dilated cardiomyopathy and depressed ejection fraction. The M/V ratio was used as representative of the relative degree of hypertrophy, and patients with an M/V ratio < 0.90 had poorer prognosis than those in excess of this value.

6.4.2 Relative wall thickness as a criterion for classification of LVH

Concentric LV hypertrophy is seen in aortic stenosis and hypertension (Badeer 1964, Linzbach 1960, Grant et al 1965, Levine et al 1963) while eccentric hypertrophy is most commonly seen in aortic and mitral regurgitation (Badeer 1964, Linzbach 1960). It has been suggested that myocardial hypertrophy may be considered as an interface between the normal and failing heart (Alpert 1971), because generally it is assumed that hypertrophy of the cardiac muscle is a useful physiological adaptation which develops when an increased work load is chronically imposed on the myocardium (1964). Grant et al (1965) examined LV chamber size and wall thickness in 5 patients with LV pressure overload and 10 patients with volume overload, and their data indicated that eccentric hypertrophy is analogous to the normal growth process by

which a neonatal left ventricle is converted into the adult ventricle by maintaining a normal relative wall thickness.

Grossman et al (1975) in a clinical angio-cardiographic study concluded that the h/R ratio (the ratio of the wall thickness h to the LV radius R) was almost similar in patients with normal LV geometry (34 ± 0.02) and those with volume overload (38 ± 0.02) in comparison to higher values (58 ± 0.05) in patients with chronic pressure overload. However, the number of the patients in each group of Grossman et al (1975) was small (normals=6, pressure overload=6 and volume overload=18) while the volume overload patients included three patients who had a normal left ventricular mass index of 86, 107 and 114 g/m^2 respectively.

6.4.3 Limitations of relative wall thickness in LVH classification

In Grossman's work (1975), h/R was not able to distinguish the three patients with volume overload, and normal LV mass and low h/R ratio from the other patients with volume overload with increased LV mass. Moreover, the actual volumes of the left ventricle were not mentioned.

In the present study, 10 patients are described who have normal LV masses with enlarged LV volumes. They have a mean RWT of 22.087 ± 4.873 and a quite distinctive LV geometry (decompensated LV) compared to the other three groups. This indicates that the only possible way to

diagnose a decompensated LV from an M-mode echocardiogram is by actual calculation of the indexed masses and volumes.

There were other limitations in utilizing the RWT for the classification of LVH in this sample of cardiovascular patients. When concentric LVH was diagnosed on the basis of $RWT \geq 45\%$ in the Framingham study (Savage 1987), three (2%) of 170 women and three (3%) of 113 men had slightly dilated left ventricles. In this study, ten (18%) out of the 54 patients diagnosed as concentric LVH on the basis of $RWT \geq 45\%$ had indexed LV volumes exceeding 90 ml/m^2 , while 20 (24%) of 82 patients with eccentric LVH had indexed LV volumes $< 90 \text{ ml/m}^2$ because of increased indexed LV masses on the basis of $RWT < 45\%$. Moreover, in the Framingham study, the eccentric non-dilated LVH (increased LV mass but with a $RWT < 45\%$ and normal indexed LVIDD) constituted the commonest type of LVH (Savage et al 1987). However, pathophysiologically, eccentric LVH occurs in response to volume overload i.e. dilated LV, while the h/R ratio remains almost normal (Ford 1976, Grossman 1975).

The findings of the present study with respect to (i) the utility of RWT, and (ii) on close inspection, results from the Framingham study, demonstrate limitations of this diagnostic echocardiographic criterion especially when a cut-off point of 45% is used in classification of LVH into dilated and non-dilated types. The main limitation of relative wall thickness has been stressed

by Gaasch (1979), namely that minor measurement errors potentially limit its extensive use in clinical cardiology.

Moreover, an applicable cut-off point in well defined groups of patients such as in those of Grossman et al (1975) or in a sample of the general population (Savage 1987) may not readily transfer into a sample of cardiac patients where LVH may be associated with, (i) deranged coronary flow due to valvular disease which in turn may affect the myocardial growth, (ii) progressive valvular disease causing progression of the hypertrophy, or (iii) acute cardiac failure where the h/R ratio is very sensitive because acute dilatation causes simultaneous increase of the radius and decrease in wall thickness (Ford 1976). In the present study, there were some patients who had mixed volume and pressure overload, others with overload and coronary artery disease and patients with LVH due to dilatation of the left ventricle after myocardial infarction.

Data from cardiac catheterization in the human on well defined pressure or volume overload conditions has revealed that the relationship of the h/R ratio to the systolic intra-ventricular pressure is nearly linear and independent of the ventricular size (Ford 1976). However the h/R ratio has not been studied in patients who have mixed pressure and volume overload, or a decompensated pressure overload. In the present study, the mixture of various cardiovascular pathological conditions

encountered in routine cardiological practice highlighted the anomalies in classifying left ventricular enlargement on the basis of RWT. Moreover, estimation of the indexed LV volumes from M-mode echocardiograms might also be affected by errors in the echo measurements, more so because the volume of the LV is derived from the cube of its internal diameter. The relative wall thickness is also liable to error in the measurement of the LV dimensions so that when the posterior wall of left ventricle is under or over estimated, this will be reflected on the internal dimension, thus seriously affecting the h/R ratio.

6.5 CONCLUSIONS

From this study of a sample of cardiac patients, it is concluded that -

- (1) A cut-off value $\geq 45\%$ for relative wall thickness (RWT) was not useful in differentiating dilated from non-dilated LVH;
- (2) RWT failed to diagnose a small group of patients with normal left ventricular masses but with enlarged LV chambers. These findings are most likely to be due to the wide range of pathological conditions involved in the study including patients who had a mixture of pressure and volume overloads;
- (3) Classification of LV enlargement on the basis of a newly proposed indexed mass/volume relationship separated hearts into four groups; normal LV geometry, concentric LVH (normal volume, increased mass); eccentric LVH

(increased volume, increased mass), and decompensated LV (normal mass, increased volume). The differences between the Framingham classification and this new classification of LVH/LV enlargement are shown in figures 6.5 and 6.6 respectively.

(4) A consequence of the above classification is that patients with a decompensated left ventricle should be omitted from further ECG-echocardiographic correlative studies of left ventricular hypertrophy/enlargement.

The new approach forms the basis of studying the electrocardiographic differences between concentric and eccentric types of LVH in a sample of the cardiac population. Another implication of this approach may be its use for prognostic purposes using the mass/volume ratio within the eccentric group.

Figure 6.5 Classification of LVH in Framingham Study

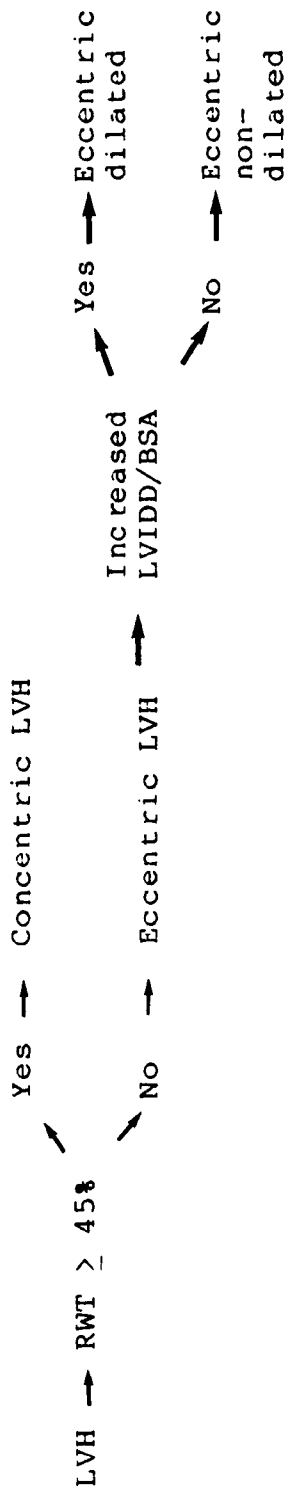
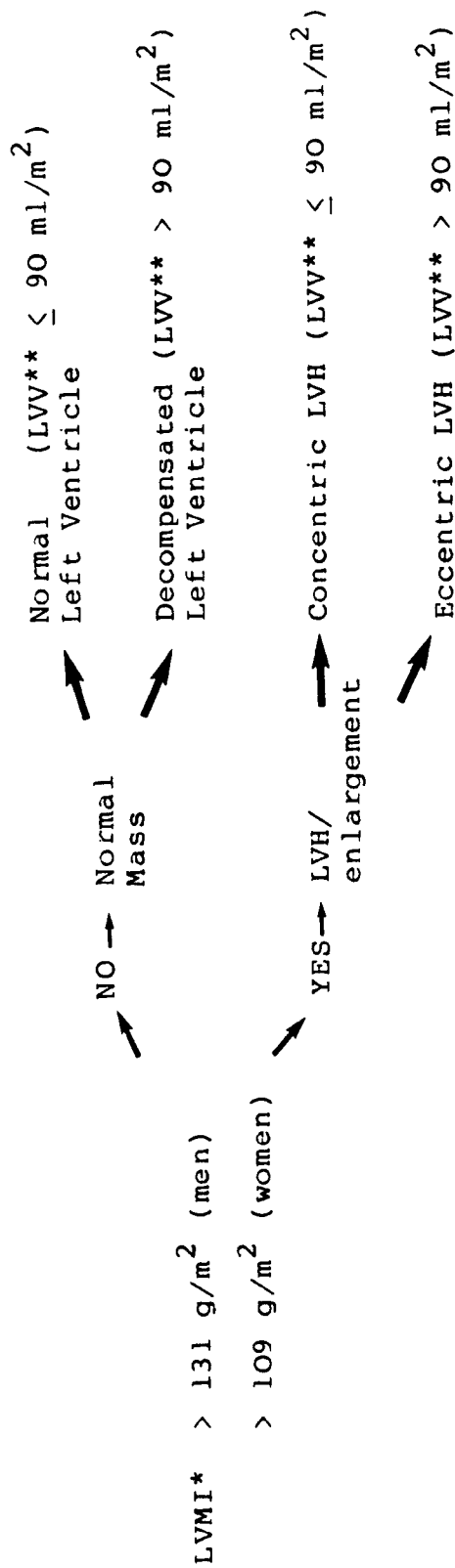


Figure 6.6 Classification of LVH/enlargement on M-mode echocardiogram



* LVMI: Left ventricular mass index calculated on Penn convention

** LVV: Left ventricular volume estimated on ASE recommendations

7. MORPHOLOGICAL PATTERNS OF ST-T ABNORMALITIES
IN LEFT VENTRICULAR HYPERTROPHY WITH NORMAL
CORONARY ARTERIES

7.1 INTRODUCTION

Carter and Estes (1964) showed that electrocardiographic left ventricular (LV) strain defined as asymmetric ST depression and T wave inversion in the anterolateral leads (I, aVL, V5 and/or V6) correlated significantly with left ventricular weight at autopsy. Others found that LV strain (ST segment depression \geq 0.1 mV with asymmetric T wave inversion) in the anterolateral leads was 95% specific for left ventricular hypertrophy (LVH) in the absence of digitalis therapy (Devereux and Reichek 1982). However, in clinical practice, it is not uncommon to observe that some patients, who have ECG-LVH confirmed by echocardiography, have symmetrical or asymmetrical T wave inversion without ST segment depression. In the Romhilt-Estes (1968) scoring system for the ECG diagnosis of LVH, points are scored for ST-T changes only if LV strain is present. On the other hand, it is not established whether T wave changes without ST-segment depression occur because of LVH per se or on account of associated coronary artery disease (CAD) particularly as it is well established that patients with ECG-LVH and ST-T abnormalities are eight times more prone to CAD than those who have ECG-LVH without repolarization wave abnormalities (Kannel, Gordon, Castelli and Margolis

1970). For these reasons it was decided to study a group of patients with echo LVH, ST and/or T abnormalities on their scalar ECG and normal coronary arteries documented by coronary angiography in order to review morphological patterns of the repolarization wave abnormalities.

7.2 PATIENTS AND METHODS

Patients were selected for this study if they had LVH, documented by echo, and normal coronary arteries. As a result, they either had aortic valve disease (AVD) or hypertensive heart disease. No patient had a previous myocardial infarction, IV conduction defect, or was receiving digitalis therapy. Myocardial infarction was excluded in these patients on the basis of clinical history and contrast left ventriculography. All patients had ECGs, echocardiograms and cardiac catheterization with coronary angiography as a part of their general assessment.

The ECGs were recorded with either a Mingorec 4 Electrocardiograph or a computer compatible Electrocardiograph developed locally (Watts and Shoat 1987). ST segment depression was regarded as present when depression of ≥ 0.1 mV occurred 40 milliseconds (msec.) after the J point. The morphology of the ST-T changes was determined according to the following rules:

- (1) ST-segment depression was classified as downward sloping, flat or upward sloping.
- (2) T wave inversion was classified as symmetrical or asymmetrical with terminal overshoot.

The M-mode echocardiograms were recorded as described earlier in Chapter 5. Measurements of the interventricular septum (IVS), posterior wall of the left ventricle (PWL) and the left ventricular internal dimension (LVID) were made according to the Penn convention (Devereux and Reichek 1977) at the tip of the mitral valve at end-diastole. The left ventricular masses (LVM) were calculated by the formula of Devereux and Reichek (1977) as follows:

$$LVM = \{(IVS + PWL + LVID)^3 - (LVID)^3\} - 13.6 \text{ gms}$$

The LVMS were indexed to the body surface area (BSA) the latter being obtained from the following equation (DuBois and DuBois 1916)

$$BSA = 0.0001 \times 71.84 \times Wt^{0.425} \times Ht^{0.725}$$

where BSA is in meter², weight (wt) in Kg and Height (Ht) in centimetres.

Echocardiographic LVH was diagnosed when the indexed left ventricular mass index (LVMI) exceeded 131 g/m² or 109 g/m² in men and women respectively (Devereux et al 1984). Absence of coronary artery disease was defined as ABSOLUTELY normal coronary arteries.

7.3 RESULTS

24 patients completed the study. They included 18 men and 6 women with a mean age of 53.2 \pm 10.46 years, range 31 to 72 years. All had echocardiographic LVH and their LVMI ranged from 137 to 307 g/m² with a mean of

$189.34 \pm 53.7 \text{ g/m}^2$. Left ventricular hypertrophy was due to hypertension in 16/24 (66%) and aortic valve disease (aortic stenosis and/or regurgitation) in 8/24 (34%) of the patients. All had absolutely normal coronary arteries. Their LVM indexed to BSA (g/m^2), QRS axes, and distribution of the pattern of the ST segment depression and T wave inversion is shown in table 7.1. This data can be summarized as follows:

- (1) Asymmetric ST-segment depression (downward sloping with $\text{ST} \geq 0.1 \text{ mV}$, 40 msec after J point) convex upward in the anterolateral leads, was observed in 17/24 (71%) of the patients. The T wave changes in these 17 patients were as follows:
 - (a) Asymmetric T waves $\leq 0.00 \text{ mV}$ in the antero-lateral leads in 14/24 (58%) of the patients as shown in figure 7.1.
 - (b) Flat T waves in 2/24 (8%).
 - (c) Symmetric T wave inversion in 1/24 (4%).
- (2) Flat ST segment depression ($\geq 0.1 \text{ mV}$, 40 msec after the J point) was found in 2/24 (8%) of the patients as shown in figure 7.2. Both these patients had asymmetric T wave inversion in the anterolateral leads.
- (3) Isolated T wave changes ($\leq 0.00 \text{ mV}$) in the antero-lateral leads were present in 5/24 (20%) of the patients. They included:
 - (a) 3/24 (12%) patients with asymmetric T wave inversions (figure 7.3).

Table 7.1 Clinical, ECG and Echo data of the patients with echo LVH and normal coronary arteries.

Name	Sex	Age (yrs)	LVM ₁ (g/m ²)	Diag.	ECG LVH\$	ST segment depression	T wave inversion	QRS axis
GG	M	58	169.6	HBP	-	I, aVL, V6*	I, aVL, V5, V6*	30
AC	F	52	127.8	"	+	I, aVL, V2-V6*	I, aVL, V2-V6**	12
SE	M	45	162.3	"	-	nil	I, aVL, V6**	16
CL	F	38	193.0	"	-	I, aVL, V5, V6*	I, aVL, V5, V6**	43
DMC	M	53	145.0	"	-	V5, V6*	aVL (flat)	35
JN	M	31	137	"	+	I, aVL, V3-V6*	I, aVL, V4-V6**	28
							V3 (symmet)	
JH	M	63	180.1	"	-	nil	aVL (flat)	66
KMC	M	62	216.0	"	+	nil	I, aVL, V6 (symmet)	36
RM	M	39	144	"	-	I, aVL, V5, V6*	I, aVL**	03
TC	M	55	245	"	-	nil	aVL**	-05
JG	M	53	145	"	+	aVL (flat)	aVL**	17
EP	F	46	126.1	"	+	V5*	aVL (flat)	67
EH	F	53	140.0	"	-	V5, V6 (flat)	I, aVL, V5, V6**	-07
JB	M	54	193.3	"	+	aVL*	I, aVL, V5, V6**	25
CW	M	52	193.0	"	-	I, aVL, V5, V6*	I, aVL, V5, V6**	14
AD	F	54	305	"	+	I, aVL, V5, V6*	I, aVL, V5, V6**	01
AB	M	56	220	AVD	+	I, aVL, V4-V6*	I, III, aVL, V4-V6**	-01
JM	M	36	155	"	-	nil	I, II, III, aVL, VF, V5, V6**	42
HS	M	72	197	"	+	I, aVL, VF, V3-V6*	I, aVL, VF, V3-V6**	04
GW	M	56	246	"	+	I, II, aVL, VF, V5 V6*	I, II, VF, V5, V6**	15
							aVL (symmet)	
DE	M	69	307	"	+	I, II, III, VF, V5 V6*	I, II, III, aVL, VF, V5, V6**	46
NI	M	75	137	"	+	I, aVL, V3-V6*	I, II, aVL, V3-V6**	64
SB	F	52	274	"	+	I, II, aVL, V5, V6*	I, II, aVL, V5, V6**	48
JF	M	63	186	"	+	I, II, III, aVL, VF, V5, V6*	I, II, III, aVL, VF, V5, V6**	-16

* Downward sloping ST depression (> 0.1 mV), 40 msec after the J point
 ** Asymmetric T wave inversion with terminal overshoot
 \$ ECG-LVH diagnosed according to the Glasgow criteria

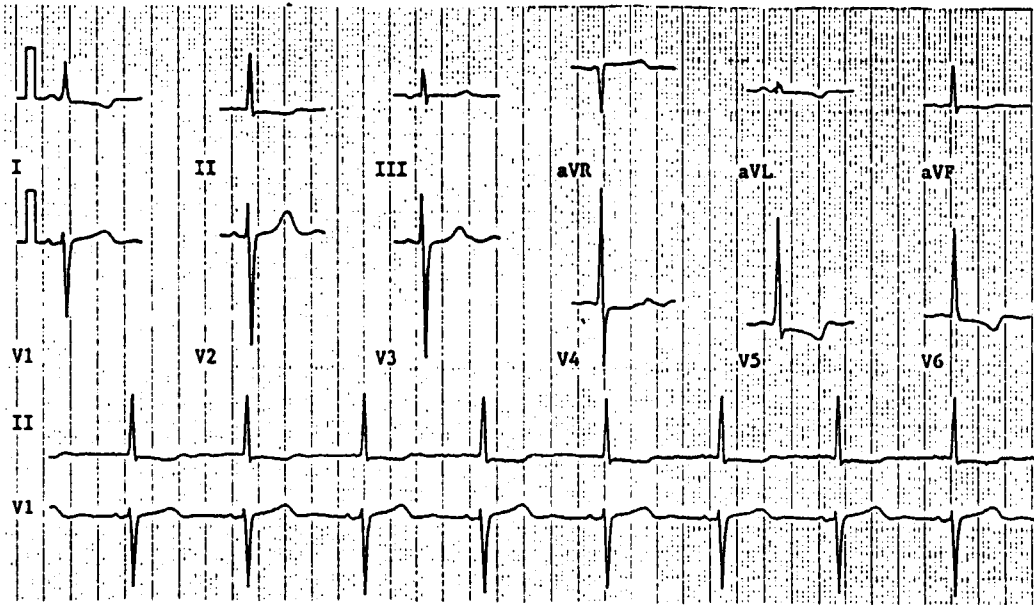


Figure 7.1

Electrocardiographic left ventricular strain. An electrocardiogram of a 52 year old female patient who had aortic valve disease with normal coronary arteries. Her echo LV mass was $274\text{g}/\text{m}^2$. The ECG shows typical left ventricular strain defined as ST segment depression (40 milliseconds after the J point) bowed upward and sloping down into an inverted T wave with terminal overshoot.

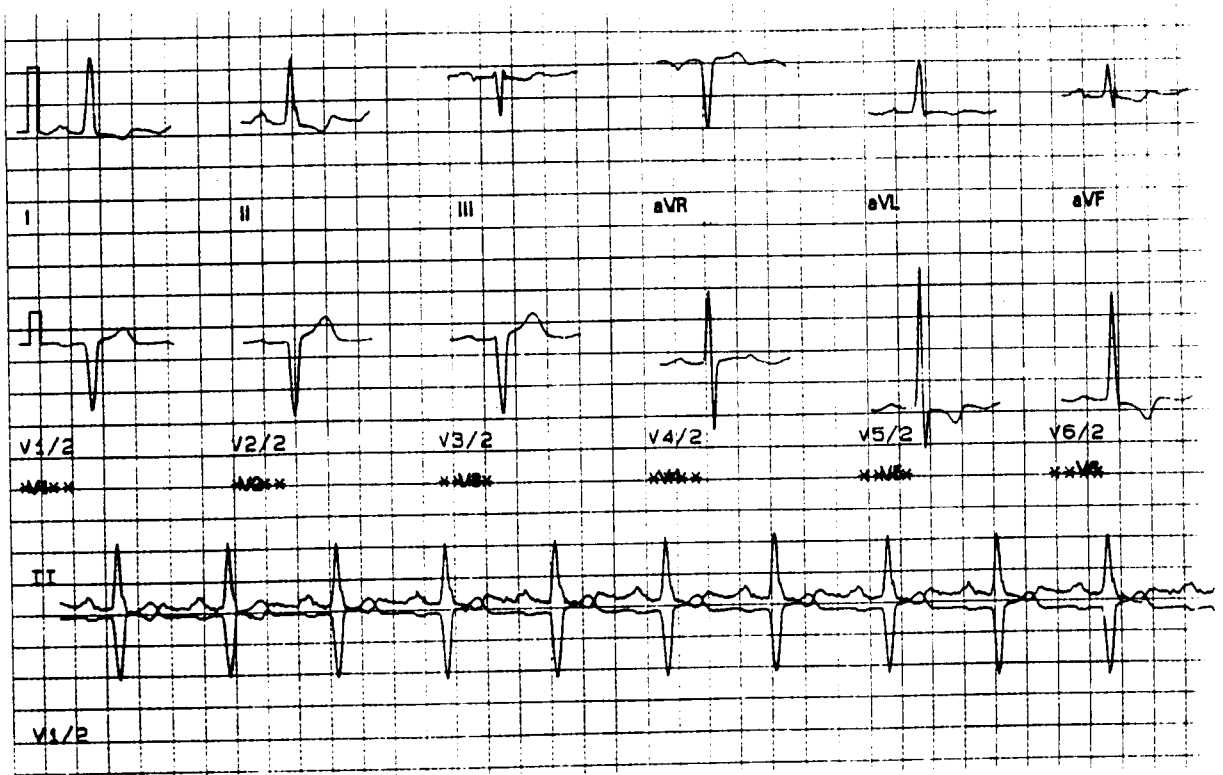


Figure 7.2

Flat ST segment depression in left ventricular hypertrophy without coronary artery disease. An electrocardiogram of a 53 year old woman with hypertension and normal coronary arteries. It shows T wave inversion in the anterolateral leads which is associated with flat ST segment depression. She had an echo LV mass of 140 g/m^2 (Penn convention).

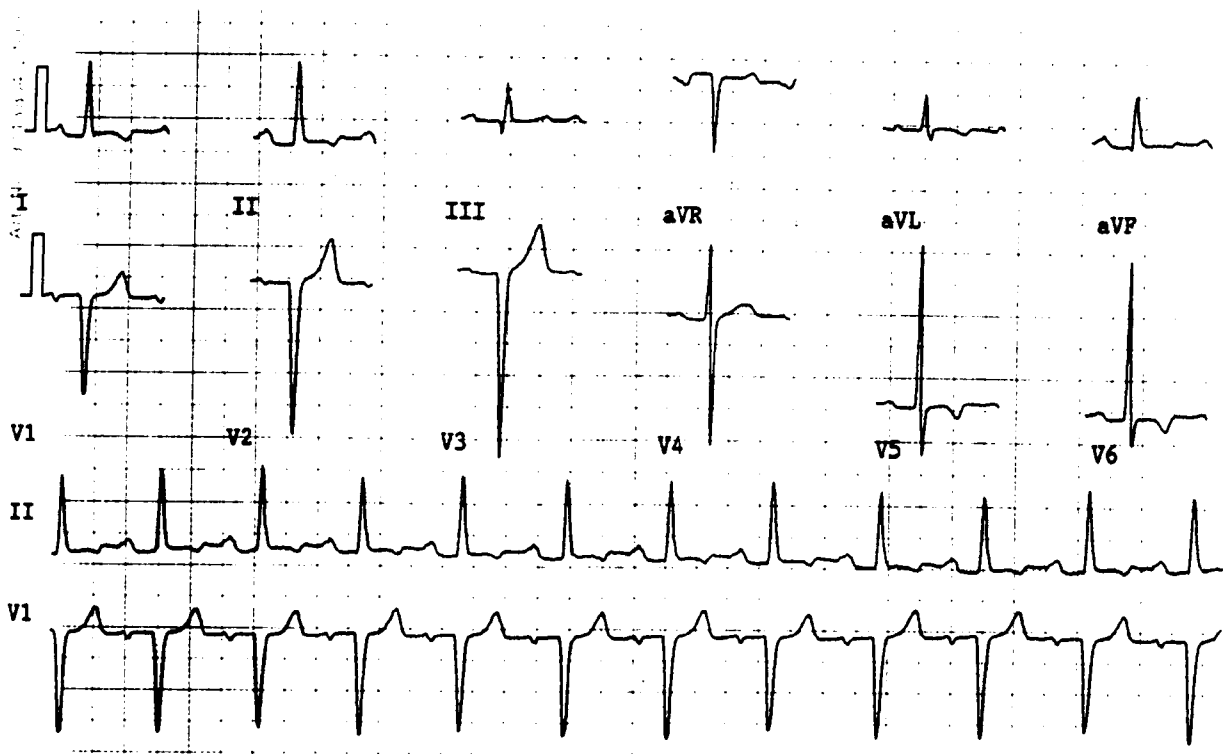


Figure 7.3

Inversion of the T waves without ST segment depression in left ventricular hypertrophy without coronary artery disease. An ECG of a 36 year old man with mixed aortic valve disease with normal coronary arteries who underwent aortic valve replacement. It shows T wave inversions in anterolateral leads without significant ST segment depression. The echo LV mass was 155 g/m^2 (Penn convention).

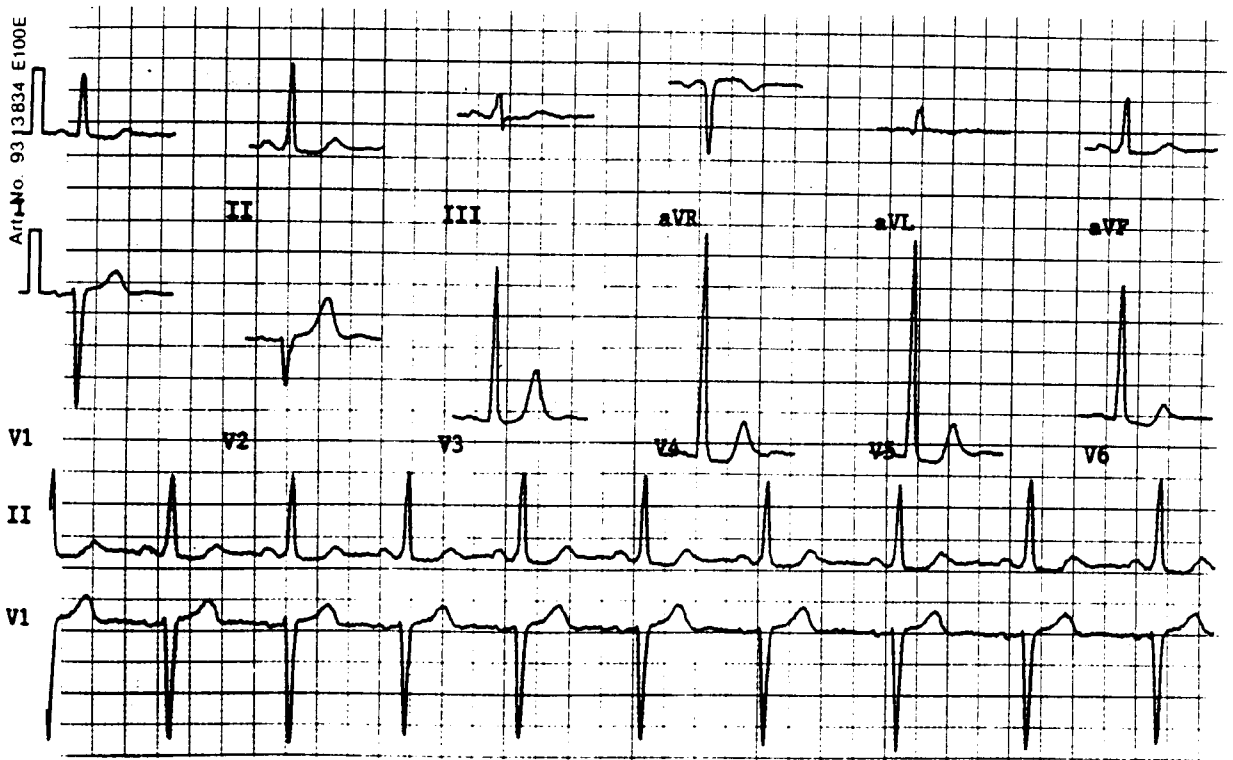


Figure 7.4

Flat T waves in left ventricular hypertrophy without coronary artery disease. An ECG of a 53 year old man with hypertension with normal coronary arteries. It shows that the T wave is flat in lead aVL while it is upright in the leads I, V5 and V6. The echo LV mass was 145 g/m^2 (Penn convention).

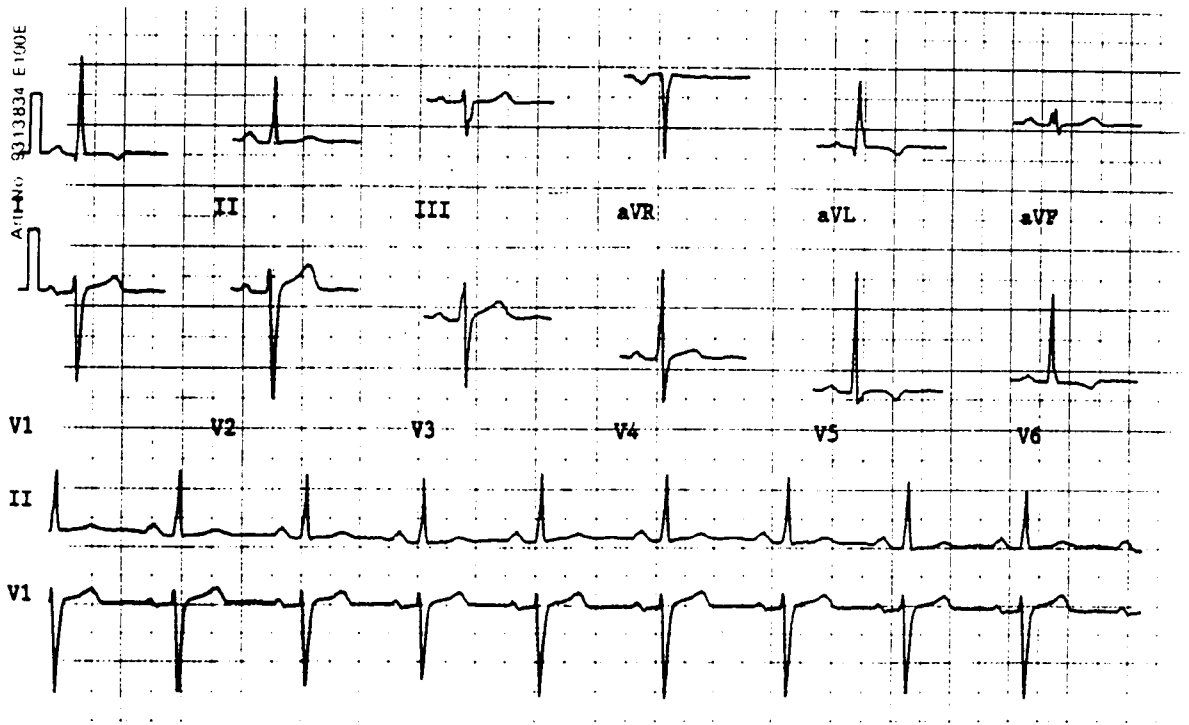


Figure 7.5

Symmetrical T wave inversion in left ventricular hypertrophy without coronary artery disease. An ECG of a 44 year old woman with hypertension with normal coronary arteries. It shows that T waves are inverted in the anterolateral leads and are symmetrical in lead V5. The echo LV mass in this patient was 162.3 g/m².

(b) 1/24 (4%) patient with flat T waves
in aVL only (figure 7.4).

(c) 1/24 (4%) patient with symmetric
T wave inversion in the anterolateral
lead V5 (figure 7.5).

7.4 DISCUSSION

The generally accepted definition of LV strain is an ST segment depression which is bowed upward and sloping down into an inverted asymmetric T wave (Milliken et al 1989) a concept which had been introduced by Rykert and Hepburn (1935). Furthermore, Beech et al (1981) suggested that repolarization wave abnormalities of LVH without CAD could be frequently differentiated from those of coronary artery disease by the presence of one or more of the following five features; depression of the J point, asymmetry of the T wave with rapid return to the baseline, terminal positivity of the T wave (overshoot), T wave inversion in V6 greater than 0.3 mV, and T wave inversion greater in V6 than in V4. However, it is not rare in clinical practice to observe ST-T abnormalities in association with LVH, which do not have the above mentioned criteria of LV strain and thus may be confused with those of coronary artery disease (CAD). The latter issue is further complicated by the fact that patients with LVH and ST-T abnormalities are more prone to CAD than those who do not have abnormal repolarization (Kannel, Gordon, Castelli and Margolis 1970).

In this study, 17/24 (71%) of the patients had

asymmetric ST segment depression (convex upward and sloping downwards), and 14/17 (82%) of these patients had asymmetric T wave inversion with terminal overshoot. However flat ST segment depression with asymmetric T wave inversion was observed in other 2/24 (8%) of these patients with LVH and normal coronary arteries. Furthermore, isolated T wave changes (flat, asymmetric or symmetric inversion) were observed in 5 out of the 24 and these constitute 20% of the repolarization wave abnormalities associated with LVH and normal coronary arteries.

The mechanism for those ST-T abnormalities which do not meet the definition of LV strain is difficult to explain but it is well established that myocardial ischemia may present on the surface ECG as flat ST segment depression, flat T waves, symmetric T inversion or non-specific ST-T abnormalities (Chou 1986b). None of these patients was on digitalis or was receiving diuretics and therefore therapy could not be blamed for the ST-T changes that did not meet the ECG criteria of LV strain. Furthermore, the absolutely normal coronary arteries documented by coronary angiography exclude coronary artery disease as a possible explanation. However, LVH with normal coronary arteries is a known cause of angina pectoris which is presumed to be due to abnormal coronary reserve which leads to relative coronary insufficiency (Cannon et al 1985). Therefore the most likely explanation for the flat ST segment

depression or isolated T wave changes in patients with LVH and normal coronary arteries is probable latent myocardial ischemia or a variant of LV strain. However a structural abnormality in the myocardium such as interstitial fibrosis as a cause of primary non-specific ST-T abnormalities in these patients with LVH and normal coronary arteries cannot be ruled out.

Finally it can be concluded that:

- (i) Typical LV strain defined as asymmetric (downward sloping) ST segment depression (≥ 0.1 mV, 40 msec after the J point) with T wave inversion occurs in approximately 70% of patients with LVH and normal coronary arteries. The T wave inversion was asymmetric (with terminal overshoot) in 82% of these patients.
- (ii) Flat ST segment depression with asymmetric T wave inversion, or isolated T wave changes can be produced in 30% of patients with LVH without CAD.

The importance of these findings will be considered later when ECG criteria for the diagnosis of LVH are discussed.

8. ELECTROCARDIOGRAPHY OF LEFT VENTRICULAR HYPERTROPHY/ENLARGEMENT

8.1 DEFINITION OF LEFT VENTRICULAR HYPERTROPHY

Left ventricular hypertrophy (LVH) is defined as left ventricular weight increased beyond the normal limits. The interventricular septum (IVS) is regarded as a part of the left ventricle (LV) and according to Bove et al (1966), pure LVH at postmortem is diagnosed when the weight of the fat free LV and IVS exceeds 200 grams provided that the weight of the free wall of the right ventricle is less than 65 grams.

Troy et al (1972) found a good correlation between left ventricular masses estimated from M-mode echocardiograms and contrast left ventriculograms. Devereux and Reichek (1977) demonstrated a good correlation between the echocardiographically (echo) estimated left ventricular mass (LVM) and the weight of the LV at postmortem.

Woythaler et al (1983) defined echocardiographic LVH (echo-LVH) as $LVM > 265$ grams (for males and females) when the LVM was calculated from LV measurements made according to the Recommendations of the American Society of Echocardiographers (ASE). The same group also showed that LVM calculated from M-mode echoes correlated better with ECG-LVH than those estimated from 2-D echoes. Moreover Devereux et al (1984) found that LVM for any subject is dependent on the body surface area (BSA).

There are significant differences between males and females with respect to LVM indexed to BSA so that echo LVH is diagnosed as $LVM > 131 \text{ g/m}^2$ in men and $> 109 \text{ g/m}^2$ in women.

The above mentioned studies show that the weight of the LV can be reliably estimated from M-mode echoes, and stratified on the basis of BSA and sex. These factors probably explain in part the differences in the QRS voltages in males and females.

8.2 ELECTROCARDIOGRAPHIC PARAMETERS USED IN THE DIAGNOSIS OF LVH

More than thirty criteria have been proposed for the diagnosis of ECG-LVH. These criteria are based on the QRS voltage in one or more of the 12 leads with or without other non-voltage criteria (Allenstein and Mori 1960). The voltage criteria mainly used include the amplitude of R waves (leads I, aVL, aVF, V4-V6) and S waves (leads III, aVR, V1-V3). The non-voltage criteria include :

- (1) P terminal force in lead V1 (Morris et al 1964);
- (2) QRS duration ≥ 0.1 second (Carter and Estes 1964);
- (3) Intrinsicoid deflection ≥ 0.05 seconds in leads V5 or V6 (Noth et al 1947);
- (4) Left axis deviation more negative than -30 degrees (Gubner and Ungerleider 1943, Carter and Estes 1964)
- (5) Asymmetrical ST segment depression and T wave inversion in leads V5 and/or V6 (Carter and Estes 1964).

However all the non-voltage criteria used in the diagnosis of ECG-LVH are affected by pathological states of the myocardium other than LVH including myocardial ischemia and infarction, valvular heart disease, myocarditis, cardiomyopathy, pericardial disease, conduction defects, electrolyte disturbances and digitalis preparations. This wide range of pathological conditions may influence ECG parameters utilized for the diagnosis of LVH, and are probably responsible for the variations of specificity and sensitivity of the ECG-LVH criteria.

8.3 EFFECTS OF CARDIAC SIZE AND THORACIC FACTORS ON THE QRS VOLTAGES

There is no argument about increased QRS voltage being due to increased cardiac mass. However, the surface ECG represents the sum of all the electrical potentials of the heart recorded at a single point at the surface of the body. Therefore, apart from the heart itself, there are other factors which ultimately affect the voltage of the ECG recorded from the surface of the body and these include:

- (1) Thoracic volume conductors
- (2) Intracardiac cavitory blood
- (3) Cardiac size
- (4) Position of the heart within the thoracic cavity

8.3.1 Thoracic volume conductors

Several structures are involved in the conduction of the myocardial potentials to the surface of the body

through the thorax. These include:

- (1) Pericardium
- (2) Lungs
- (3) Skeletal muscles
- (4) Subcutaneous fat

Although the passage of the electrical potentials in the thoracic volume conductors is passive, the structures that constitute that volume have different resistivities and conductivities (Rudy 1987).

The blood has the highest conductivity compared to the myocardium and the other intra-thoracic structures. The skeletal muscles are good conductors of electrical current in contrast to the pericardium, lungs and the subcutaneous fat (Rudy 1987). This explains why voltage is increased after mastectomy, but is reduced in patients with obesity, emphysema and pericardial disease.

8.3.2 Intracardiac cavitory blood

The presence of blood in a spherical cavity surrounded by the myocardium theoretically enhances potentials due to radial excitation and attenuates those due to excitation tangential to the blood cavity (Brody 1956). Therefore augmentation of the surface potentials following an increase in the volume of the blood inside the heart is expected. This is known as the Brody effect. Moreover haemoglobinization of the blood also affects the blood conductivity so that anaemia causes increased voltage, as has also been observed in patients with polycythemia when the haematocrit had been reduced

(Rosenthal et al 1971).

8.3.3 Cardiac size

The effects of the cardiac size on the QRS voltage have been studied with conflicting results. Manoach et al (1971, 1972a, 1972b) found in the normal cat that reduction of the intracavitary blood and volume by clamping the inferior vena cava caused reduction of the voltage which was restored when volume returned to normal. An increase of QRS voltage occurred when the blood volume in the left ventricle was increased by clamping the aorta or by overfilling the left ventricle. However Battler et al (1980), in the conscious dog, observed an inverse relationship between the cardiac size and the surface QRS complex voltage.

Similar results have been found in the human by Ishikawa et al (1971) in patients with a dilated heart due to congestive cardiac failure. They observed an increase of the QRS voltage when the cardiac volume was reduced. However, it has been shown in experimental models and humans that fluid in the lungs cause a reduction of the QRS voltage (Rudy et al 1982), and therefore pulmonary congestion in cardiac failure presumably causes reduction of the QRS voltages in association with dilated ventricles.

In order to clarify the above mentioned contradictory effects of cardiac size on the QRS voltage, Rudy (1987) developed an experimental model with which he showed that increased LV wall thickness and normal or augmented LV

cavity size caused increased QRS voltages. However, he also found in his experimental model that increased LV wall thickness associated with reduction of LV cavity size, resulted in reduction of the QRS voltages.

8.3.4. Position of the heart within the thoracic cavity

The position of the heart within the thoracic cavity affects the surface voltage as was proved in Rudy's experimental models (Rudy 1987). The abnormal position of the heart within the thoracic cage in patients with kyphoscoliosis for example makes the interpretation of conventional voltage criteria for ventricular hypertrophy unreliable.

8.4 PATHOPHYSIOLOGIC TYPES OF LEFT VENTRICULAR HYPERTROPHY/ENLARGEMENT

It is generally assumed that hypertrophy of the cardiac muscle is a useful physiological adaptation which develops when an increased workload is chronically imposed on the myocardium (Badeer 1964). Concentric LVH occurs in response to chronic pressure overload such as in aortic stenosis and hypertension while eccentric LVH occurs due to chronic volume overload (Braunwald 1980). The basic geometric difference between these two types of LVH is that in concentric LVH there is increased wall thickness with the LV cavity size being normal or reduced, while in eccentric LVH the increased LV internal dimension (and hence volume) stimulates the increased LV mass. Grossman (1975) suggested that eccentric LVH occurs in a way which is analogous to the normal growth

by which a neonatal ventricle develops into an adult ventricle. Moreover according to Brody (1956) and Rudy (1987), the increased intra-cardiac blood volume causes increased QRS voltage. Therefore, it is not clear how this difference in LV volume in concentric and eccentric LVH influences those ECG parameters used in the ECG diagnosis of LVH, i.e. ECG-LVH criteria.

8.5 ELECTROCARDIOGRAPHIC CONCEPTS OF SYSTOLIC AND DIASTOLIC OVERLOAD

Cabrera and Monroy (1952) introduced the terms systolic and diastolic overload for differentiating the left ventricular hypertrophy which occurs due to pressure overload from that which results from volume overload. Systolic overload may be due to aortic stenosis, systemic hypertension or coarctation of the aorta, while diastolic overload may be due to mitral or aortic insufficiency, ventricular septal defect or patent ductus arteriosus. Cabrera (1952) suggested that asymmetrical ST-segment depression and T wave inversion in leads V5 and V6 is characteristic of systolic overload conditions of the heart, while diastolic overload produces tall upright T waves in the left precordial leads in association with deeper Q waves and taller R waves in the same leads. However, the electrocardiographic concepts of systolic and diastolic overload have not previously been evaluated in relation to echocardiographically determined left ventricular mass and volume.

8.6 Q-WAVE ANTEROSEPTAL MYOCARDIAL INFARCTION AND ECG-LVH

Poor progression of the R wave in the right and mid precordial leads is frequently noted in patients with left ventricular hypertrophy. Occasionally the R waves are absent in leads V1, V2 and even lead V3 resulting in QS deflections in these leads which mimic anteroseptal myocardial infarction (Chou 1986a) as shown in figure 8.1. Levy et al (1988) showed that both angina pectoris and myocardial infarction are independent predictors of left ventricular hypertrophy. Furthermore, it is theoretically possible that the deep QS deflections in V1-V4 due to anteroseptal myocardial infarction may produce tall R waves in leads V5-V6 due to the effect of lack of cancellation of oppositely directed electrical forces and thereby may cause false positive precordial voltage criteria of LVH. Therefore, the effects of the QS deflections in leads V1-V4 on the ECG diagnosis of LVH need to be clarified. For this reason, echo left ventricular masses require to be studied in patients who have QS waves in leads V1-V4 with or without anteroseptal myocardial infarction. This makes it possible to assess the effects of anteroseptal MI on (i) precordial voltages, (ii) limb lead voltages, and hence the specificity and sensitivity of ECG-LVH criteria, including those from Glasgow.

8.7 PROGNOSTIC SIGNIFICANCE OF LVH AND LV DILATATION

The prevalence of cardiac enlargement on a chest

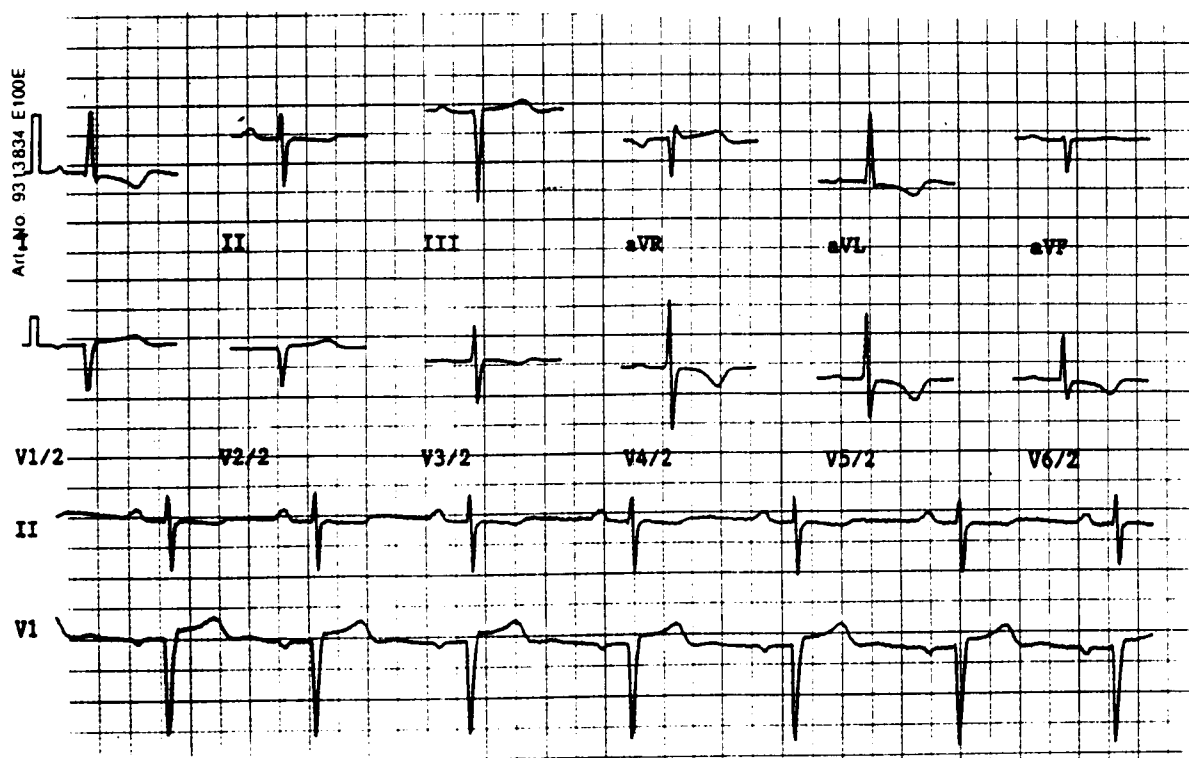


Figure 8.1

ECG of pseudo MI in a patient with left ventricular hypertrophy. Electrocardiogram of a 54 year old woman with hypertension who had a normal Thallium scan and normal coronary arteries. It shows QS deflections in leads V1 and V2 (pseudo-myocardial infarction). The echo left ventricular mass was 305 g/m₂ (Penn convention).

x-ray is approximately double that of ECG-LVH for any given age while only 16% of those patients with radiographic cardiomegaly develop ECG-LVH over a decade of follow-up (Kannel et al 1969). Radiographically determined LV dilatation has been shown to be associated with increased risk of coronary artery disease, cardiovascular mortality, or both, in clinical population samples (Hammermeister, Chikos, Fisher and Dodge 1979, Christie and Gardner 1979, Casale et al 1986).

In the Framingham study, ECG-LVH diagnosed on the basis of both voltage criteria and ST-T abnormalities, carried an eightfold increase in cardiovascular mortality and a sixfold increase in coronary artery disease mortality (Kannel et al 1981). Angina pectoris, myocardial infarction and sudden death occurred with about the same frequency in asymptomatic patients with ECG-LVH and ST-T abnormalities as in recognized victims of myocardial infarction (Kannel et al 1981).

ECG-LVH with ST-T abnormalities preceded 27% of overt coronary artery disease in the Framingham study (Kannel, McNamara and Feinleib 1970), possibly reflecting a state of silent asymptomatic coronary artery disease. Moreover, non-specific ST-T wave abnormalities alone were not associated with any greater risk than that associated with voltage electrocardiographic LVH alone (Kannel et al 1981).

8.8 COMPUTERIZED INTERPRETATION OF THE ECG IN THE DIAGNOSIS OF LVH

There is no doubt that computerized interpretation of the ECG allows accurate measurements of the amplitudes, durations and axes of the ECG waves. Moreover, the technique also offers other potential advantages in the field of ECG diagnosis of LVH which are normally precluded by the time available to a busy clinician reporting ECGs. These include the following:

- (1) Comparison of the QRS voltages with age and sex based normal limits. The effects of age and sex on QRS voltage have been demonstrated in Glasgow in a sample of over 1300 apparently healthy subjects (Macfarlane and Lawrie 1989a). It was found that QRS voltage increases with age towards 30 years of age and then begins to decrease thereafter. In addition, there are significant differences between sexes in early life although these tend to disappear by the age of 50 years. The values used in the Glasgow ECG analysis program as upper limits of normal voltages are shown in table 8.1
- (2) Application of the modified point scoring system initially designed by Romhilt and Estes (1968) i.e. the Glasgow criteria (Macfarlane 1987)
The Romhilt-Estes point scoring system is composed of a combination of voltage and non-voltage criteria so that definite LVH is diagnosed when the patient has 5 points while a score of 4

Table 8.1 Values used in Glasgow ECG analysis program as the upper limits of normal voltage for use in Romhilt-Estes point score system (Macfarlane and Lawrie 1989).

Age(years)	Sex	Lead Voltage (mV)				Lewis index
		RI	RaVL	SV1/SV2	RV5/RV6	
17-29	Male	1.5	1.1	4.0	4.0	2.5
	Female	1.5	0.9	3.5	2.5	2.0
30-39	Male	1.6	1.2	3.5	3.0	2.5
	Female	1.4	1.0	3.0	2.2	1.8
40-49	Male	1.6	1.3	2.5	2.5	2.0
	Female	1.4	1.2	2.5	2.2	1.8
> 50	Male	1.6	1.3	2.0	2.5	2.0
	Female	1.4	1.2	2.0	2.2	1.8

points is compatible with the diagnosis of probable LVH (table 8.2).

- (3) Utilization of regression equations derived from ECG parameters to predict LVH and LV mass index. Casale et al (1987) showed that multiple logistic regression exponents derived for detection of ECG-LVH achieved a higher predictive accuracy than the Sokolow-Lyon and the Cornell voltage criteria. Rautaharju et al (1988) obtained regression equations for the prediction of left ventricular mass index (LVMI) from the ECG. Left ventricular mass index obtained from those regression equations correlated well with echo derived LV masses. Moreover, these authors also observed that increased LVMI identified a substantially larger fraction of persons at increased risk of cardiovascular mortality than the conventional ECG-LVH criteria.

8.9 CORRELATIVE STUDY OF THE ECG AND ECHO LV MASS/VOLUME

A study was designed to correlate computerized 12 lead ECGs with left ventricular masses and volumes estimated from M-mode echocardiograms for the following purposes:

- (1) Evaluation of the commonly used ECG-LVH criteria in comparison with the Glasgow criteria in a population of cardiac patients with and without myocardial infarction (MI).

Table 8.2 Romhilt Estes point score system (1968)

Criteria	Points

(1) Any limb lead R or S > 2.0 mV or SV1 or SV2 ≥ 3.0 mV or RV5 or RV6 ≥ 3.0 mV	3
(2) ST-T vector shifted in direction opposite to the mean QRS	
a. With digitalis	1
b. Without digitalis	3
(3) Left atrial overload, i.e. abnormal P terminal force in lead V1 ≥ 4.0 mV.ms.	3
(4) Left axis deviation of -30 degrees or more	2
(5) QRS duration ≥ 0.09 second	1
(6) Intrinsicoid deflection in leads V5 or V6 ≥ 0.05 second	1

4 points = possible LVH

5 points = probable LVH

≥ 6 points = definite LVH

- (2) Correlation of indexed left ventricular mass with
(a) voltage criteria (R aVL, SV1, SV2, SV3, RV5, RV6, SV1 + RV5, and SV3 + R aVL), (b) non-voltage criteria utilized in the diagnosis of ECG-LVH, and (c) the LVH point scores of the Glasgow criteria.
- (3) Assessment of the multiple logistic regression exponent for detection of ECG-LVH (Casale et al 1987) and the regression equation for the prediction of left ventricular mass index (Rautaharju et al 1988).
- (4) Differentiation of concentric and eccentric types of LVH using the scalar electrocardiogram.
- (5) To study the effects of Q waves in leads V1-V4 on the precordial and limb lead voltage criteria, and the Glasgow point scoring system for the ECG diagnosis of LVH.

All patients had conventional 12 lead ECGs recorded by a Siemens Mingorec 4 electrocardiograph or by a computer compatible electrocardiograph developed locally (Watts and Shoat 1987). All the amplitudes, durations and axes of the ECG waves were measured by computer using a program described elsewhere (Macfarlane et al 1986). The ECGs were also checked manually to avoid possible computer errors. All the ECGs were recorded within three months of the M-mode echocardiographic recordings, which were obtained as described in chapter 5.

Consecutive patients undergoing echocardiography for

investigation of cardiovascular diseases were considered for the study. Only patients with echocardiograms of good quality were included. Exclusion criteria were as follows:

- (1) Conduction defects such as right and left bundle branch blocks (RBBB and LBBB), as well as intra-ventricular conduction defects (IVCD).
- (2) Wolff Parkinson White syndrome (WPW).
- (3) Pericardial disease
- (4) Hypertrophic obstructive cardiomyopathy (HOCM).
- (5) Negroid patients because of their small number.
- (6) Echocardiographic decompensated left ventricle i.e. those patients who had a normal indexed left ventricular mass but an increased left ventricular volume.

Furthermore the patients included in the study were classified into two groups:

- (i) Group 1 included all the patients who had no previous myocardial infarction. The various ECG-LVH criteria, the effects of LV geometry thereon, ECG-LVH criteria, the regression equations for the detection of LVH and the prediction of left ventricular mass index were studied in this group.
- (ii) Group 2 comprised of patients who had sustained a previous antero-septal myocardial infarction. The effects of the QS deflections in leads V1-V4 on the specificity

and sensitivity of the ECG-LVH criteria were studied in this group, which was also compared with another group of patients with pathological Q waves in leads V1-V4 but with echocardiographically documented LVH due to causes other than coronary artery disease.

8.10 CLINICAL AND ECHO DATA OF PATIENTS WITHOUT MYOCARDIAL INFARCTION

157 consecutive cardiac patients were considered for this part of the study. All had adequate M-mode echocardiograms from which left ventricular volumes and masses were calculated. 28 patients were excluded from the study because 8 had LBBB, 3 had RBBB, 2 had WPW syndrome, 8 had an old myocardial infarction while there were 7 patients with dilated left ventricles but with normal LV masses. As a result, 129 patients had satisfactory ECGs and echocardiograms for a comparative study. They included 73 males and 56 females with a mean age of 58.3 years and an age range of 19-78 years. The underlying clinical conditions included hypertension, valvular heart disease, coronary artery disease (CAD), congestive cardiac failure etc. (table 8.3)

Echo LVH was defined as $LVM \geq 132 \text{ g/m}^2$ and $\geq 109 \text{ g/m}^2$ in men and women respectively (Devereux et al 1984). However for multiple linear regression analysis in which sex is incorporated into the equation, LVH is defined as $LVM > 125 \text{ g/m}^2$ in both sexes (Casale et al 1985). LV dilatation was defined as $LVV > 90 \text{ ml/m}^2$ when the LVIDD

Table 8.3 Underlying clinical conditions of the
 patients included in the ECG-ECHO
 correlation (population of patients
 without MI).

Diagnosis	Number of patients

(1) Hypertension	41
(2) Aortic valve disease	35
(3) Mitral valve disease	25
(4) Aortic and mitral valve disease	07
(5) Aortic valve disease and coronary artery disease	02
(6) Congestive cardiac failure of unknown cause	05
(7) Others	<u>14</u>
	129

was measured according to the ASE recommendations.

The patients were classified into three groups according to their indexed left ventricular masses and volumes as described in chapter 6 and the results were as follows:

- (i) Group 1 consisted of 45 patients who had normal left ventricular volumes and masses (27 men, 18 women, with a mean age of 49.1 ± 11.8 years and age range of 19-72 years).
- (ii) Group 2 consisted of 43 patients with concentric LVH (LVH with $LVM \leq 90 \text{ ml/m}^2$). They included 22 men and 21 women with a mean age of 57.3 ± 12.2 years and an age range of 32-78 years.
- (iii) Group 3 consisted of 41 patients with eccentric LVH (LVH with $LVM > 90 \text{ ml/m}^2$). There were 24 men and 17 women with a mean age of 56.8 ± 14.06 years and age range of 20-75 years.

The mean \pm SD and the range of the indexed LV masses and volumes of the population of patients studied are shown in table 8.4. Furthermore, severe LVH defined as $LVM > 208 \text{ g/m}^2$ and $> 163 \text{ g/m}^2$ in men and women respectively, was found in 37 (45%) of the 81 patients with echo-LVH.

8.11 CORRELATION OF LEFT VENTRICULAR MASSES AND ECG PARAMETERS IN PATIENTS WITHOUT MYOCARDIAL INFARCTION

The following ECG parameters were correlated with LVM estimated from M-mode echocardiograms in patients

Table 8.4 Mean \pm SD and range of the indexed LVV and LVM of the three groups of the patients included in the study.

Group	No. of		LVV/BSA	LVM/BSA
	patients		ml/m ²	g/m ²

"Normals"	45	mean \pm SD	57.6 \pm 17.7	97.58 \pm 17.7
		range	27-87	64-130
Concentric	43	mean \pm SD	56.9 \pm 17.2	182.53 \pm 58.2
		range	17-88	116-339
Eccentric	41	mean \pm SD	145.7 \pm 49.39	207.67 \pm 65.6
		range	91-315	120-355

without myocardial infarction:

- (1) Amplitude of R in lead aVL in males and females.
- (2) Amplitude of S in lead V3 in both sexes.
- (3) Sum of the amplitude of SV3 and R aVL in men and women.
- (4) Amplitudes of SV2, RV5, RV6 and SV1 + RV5.
- (5) Non-voltage ECG parameters used in the diagnosis of LVH, namely P terminal force in lead V1, QRS duration, intrinsicoid deflection in leads V5 and V6, non-specific ST-T abnormalities (ST-segment depression with or without T wave inversion in leads V5 and/or V6 of any magnitude), and positive T wave amplitude in lead V1. Non-specific ST-T abnormalities in leads V5 and/or V6 were studied because they were utilized in the Framingham study for the assessment of prognosis of patients with ECG-LVH (Kannel et al 1981).
- (6) LVH score of the old Glasgow point scoring system (Macfarlane and Lawrie 1989b).

The non-voltage criteria which had good correlation with the indexed left ventricular masses independently included QRS duration ($r = 0.3167$), intrinsicoid deflections in leads V5 and V6 ($r = 0.3168$ and $r = 0.2452$ respectively), and positive T-wave amplitude ($r = 0.3708$). However, the P terminal force in lead V1 had a poor correlation ($r = 0.159$) with the indexed left ventricular masses (table 8.5). A poor correlation was also found between the indexed LV mass and non-specific

ST-T depression in leads V5 and V6 ($r = 0.0934$ and 0.0111 respectively).

There was a weak positive correlation ($r = 0.1855$) with $p < 0.05$ between the amplitude of the S wave in lead V3 and echo LVM. On the other hand, the amplitude of the R wave in V5 also did not correlate well ($r = 0.0995$) with the echo LVM, although the R amplitude in lead V6 correlated a little better ($r = 0.1813$). Although there was a poor correlation between echo LVM and the amplitude of the R wave in lead V5, the sum of SV1 + RV5 correlated better ($r = 0.2015$).

The amplitude of SV3 had a poor correlation with the indexed LV mass in women ($r = -0.0338$) but had a good correlation in men with $r = 0.3177$ (table 8.5). Moreover, the amplitude of the R wave in lead aVL showed a good correlation with the indexed LV mass in females ($r = 0.4833$) but a poor correlation in males ($r = 0.118$). The sum of the amplitudes of SV3 and R aVL correlated well with the indexed left ventricular masses in both males and females with r values of 0.3548 ($p < 0.05$) and 0.2696 ($p < 0.05$) respectively (table 8.5).

Moreover, there was a positive correlation ($r = 0.3871$) between the echo LVM and the LVH score derived from the old Glasgow scoring system.

8.12 ANALYSIS OF THE ECG CRITERIA USED IN THE DIAGNOSIS OF LVH WITHOUT MI

ECG-LVH criteria were assessed in relation to the echocardiographically derived LV masses with respect to

Table 8.5 Correlations of indexed left ventricular mass with selected ECG parameters.

ECG Parameters	(r) value	p value
P terminal force in lead V1*	0.159	0.0987
QRS duration	0.3167	< 0.05
Intrinsicoid deflection		
Lead V5	0.3168	< 0.05
Lead V6	0.2452	< 0.05
Positive T wave amplitude V1	0.3708	< 0.05
Non-specific ST-T depression		
Lead V5	0.0934	0.2894
Lead V6	0.0111	0.2042
Amplitude of SV2	0.1885	< 0.05
Amplitude of SV3		
Men	0.3177	< 0.05
Women	- 0.0338	0.8619
Amplitude of R aVL		
Men	0.118	0.3430
Women	0.4833	< 0.05
Amplitude of SV3 + R aVL		
Men	0.3548	< 0.05
Women	0.2696	< 0.05
Amplitude of RV5	0.0995	0.2589
Amplitude of RV6	0.1813	< 0.05
SV1 + RV5	0.2013	< 0.05
LVH score (old Glasgow criteria)	0.3871	< 0.001

* The correlation of the indexed left ventricular mass with the ECG parameters was made in 129 patients except for the P terminal force in lead V1 which was made in 79 patients only.

their sensitivity and specificity. All the criteria were tested after exclusion of the patients who had a history of myocardial infarction. The ECG-LVH criteria analysed included the following:

- (1) $SV_1 + RV_5 \text{ or } RV_6 \geq 3.5 \text{ mV}$ (Sokolow and Lyon 1949).
- (2) $SV_1 + RV_5 \text{ or } RV_6 \geq 3.5 \text{ mV}$ plus non-specific ST-segment depression and T wave inversion in leads V_5 and V_6 .
- (3) $SV_1 + RV_5 \text{ or } RV_6 \geq 3.5 \text{ mV}$ plus asymmetrical ST-depression and T wave inversion. The asymmetrical ST-T changes of LV strain were defined in the old Glasgow point scoring system for the diagnosis of ECG-LVH as T wave inversion $< -200 \text{ uV}$ with ST segment depression which is downward sloping, i.e. $ST < -20 \text{ uV}$ with a slope < -5 degrees, or $ST < -50 \text{ uV}$ with a slope < 0 degrees.
- (3) $R_{aVL} + SV_3 > 3.5 \text{ mV}$ in men and $> 2.5 \text{ mV}$ in women (Casale et al 1985).
- (4) $R_{aVL} + SV_3 > 2.2 \text{ mV}$ in men or $> 1.2 \text{ mV}$ in women plus $T_{V_1} > 0 \text{ mV}$ or $> 0.20 \text{ mV}$ in patients ≤ 40 or > 40 years respectively (Casale et al 1985).
- (5) $R_{aVL} + SV_3 > 2.8 \text{ mV}$ or $> 2.0 \text{ mV}$ in men and women respectively (Casale et al 1987).
- (6) Voltage criteria used in the Romhilt-Estes point scoring system, i.e. $SV_1 \text{ or } SV_2 \geq 3.0 \text{ mV}$, $RV_5 \text{ or } RV_6 \geq 3.0 \text{ mV}$, and R in lead I or $aVL \geq 2.0 \text{ mV}$.
- (7) Non voltage criteria (P terminal force in lead $V_1 \geq 4.0 \text{ mVmsec}$, QRS duration > 0.1 second,

intrinsicoid deflection in lead V5/V6 > 0.05 secs, left axis deviation more negative than -30 degrees, non-specific ST-segment depression and T wave inversion in leads V5 and V6, ST-segment depression < -20 uV with ST slope < -5 degrees with $T < -200$ uV, and ST-segment < -50 uV with ST slope < 0 degrees and $T < -200$ uV.

- (8) Romhilt-Estes point scoring system for the diagnosis of ECG-LVH (table 8.2).
- (9) Glasgow point scoring system for the diagnosis of ECG-LVH (Macfarlane 1987). This is a modification of the Romhilt-Estes point scoring system (appendix 1).

8.13 SPECIFICITY AND SENSITIVITY OF THE ECG-LVH CRITERIA IN PATIENTS WITHOUT MYOCARDIAL INFARCTION

The voltage criteria which were highly specific for LVH were R in lead I and R aVL ≥ 2.0 mV (97.9%), RV5 or RV6 ≥ 3.0 mV (95.6%) and SV1 or SV2 ≥ 3.0 mV (97.9%). However, they had unacceptably low sensitivities of 6.2%, 13.6% and 14.8% respectively (table 8.6). The Cornell voltage criteria (R aVL + SV3 > 2.8 mV in men and > 2.0 mV in women) had a sensitivity of 72.9% but a specificity of only 53%. The first ECG-LVH criterion described by Casale et al (1985) of R aVL + SV3 > 3.5 mV in men or > 2.5 mV in women had a specificity of 89% but with a sensitivity of only 33.3%. The widely used Sokolow-Lyon criteria of SV1 + RV5 or RV6 ≥ 3.5 mV was only 58%

Table 8.6 Specificity and sensitivity of ECG-LVH
voltage criteria analysed in the
population studied.

Criteria	Sensitivity	Specificity

(1) SV1 or SV2 \geq 3.0 mV	14.8%	97.9%
(2) RV5 or RV6 \geq 3.0 mV	13.6%	95.8%
(3) RI or RaVL \geq 2.0 mV	6.2%	97.9%
(4) RaVL + SV3 > 3.5 mV and 2.5 mV in men and women respectively	33.3%	89.6%
(5) RaVL + SV3 > 2.8 mV in males and > 2.0 mV in females (Casale et al 1987)	72.9%	53.0%
(6) SV1 + RV5 or RV6 \geq 3.5 mV	59.0%	64.4%

specific for LVH with a sensitivity of 64.4% (table 8.6).

The non-voltage criteria which were highly specific for LVH included intrinsicoid deflection > 0.05 seconds in leads V5 or V6, and ST-T changes as described above. The specificity of the above mentioned three criteria were 97.9%, 89.8% and 93.7% with a low sensitivity of 9.9%, 35.8% and 19.8% respectively. The remaining non-voltage criteria, namely, P terminal force in lead V1 ≥ 4.0 mV.ms, QRS duration > 100 m.sec., non-specific ST-segment depression and T wave inversion in leads V5 and/or V6, and positive T wave amplitude in lead V1, had low specificities of 81.3%, 68.7%, 25.0% and 54.2% respectively (table 8.7).

The ECG criteria which were based on the combination of voltage and non-voltage criteria also had variable specificities and sensitivities (table 8.8). The criterion with the highest specificity was $SV_1 + RV_5$ or $RV_6 \geq 3.5$ mV plus ST-segment depression of < -50 uV and ST slope < 0 degrees with T wave inversion < -200 uV. The latter criterion has a specificity of 95.8% with a sensitivity of 19.8%. However when the Sokolow-Lyon criterion was combined with ST depression of < -20 uV and a slope of < -5 degrees with T wave inversion of < -200 uV, the specificity reduced to 91.7% but the sensitivity increased to 32.1%. The specificity of the Sokolow-Lyon criterion with the addition of non specific ST-T changes in V5 and/or V6 resulted in an unacceptable specificity

Table 8.7 Specificity and sensitivity of the individual non-voltage ECG criteria used in the diagnosis of LVH.

Criteria	Sensitivity	Specificity
(1) P terminal force in lead V1 (amplitude x duration) \geq 4.0 mV.msec	25.9%	81.3%
(2) P terminal force in lead V1 (amplitude < -100 uV plus duration \geq 40 msec)	17.9%	91.1%
(3) QRS duration \geq 0.1 sec	44.4%	68.7%
(4) Intrinsicoid deflection in leads V5 or V6 \geq 0.05 second	09.9%	97.9%
(5) Left axis deviation < -30 degrees	09.1%	96.4%
(6) Non-specific ST depression and T wave T inversion in V5 and/or V6	42.0%	25.0%
(7) ST-depression < -20 uV and slope < -5 degrees with T wave inversion < -200 uV in I, V5 or V6	35.8%	89.8%
(8) ST depression < -50 uV and slope < 0 degrees with T wave inversion < -200 uV in I, V5 or V6	19.8%	93.7%
(9) T-wave amplitude in V1 \geq 0.00 mV and > 0.20 mV (age \leq 40 and > years respectively)	58.0%	54.2%

Table 8.8 Specificity and sensitivity of combination of voltage criteria and non-voltage criteria for the diagnosis of ECG-LVH.

Criteria	Sensitivity	Specificity	Assoc.* Index
(1) SV1 + RV5 > 3.5 mV with non-specific ST segment depression and T wave inversion	49.0%	83.0%	32
(2) SV1 + RV5 > 3.5 mV with ST depression < -20 uV and slope < -5 degrees with T wave inversion < -200 uV	32.1%	91.7%	24
(3) SV1 + RV5 or RV6 > 3.5 mV with ST depression < -50 uV and slope < 0 degrees and T wave inversion < -200 uV	19.8%	95.8%	16
(4) RaVL + SV3 > 2.8 mV in men and > 1.2 mV in women with T amplitude in V1 > 0.00 mV (< 40 years) and > 0.2 mV (> 40 years)	46.9%	77.1%	24
(5) Romhilt-Estes	40.7%	81.2%	22
(6) Glasgow criteria (old system) (definite LVH > 6 points)	46.8%	91.7%	39
(7) Glasgow criteria (old) minus PTF in V1 (amplitude x duration)	32.1%	93.3%	25
(8) Glasgow criteria (old) plus Casale 1985 SV3 + RaVL > 3.5 mV in men and > 2.5 mV in women	55.9%	84.4%	40
(9) Glasgow (old) criteria plus Cornell	61.9%	75.5%	38

* Association Index = Sensitivity + Specificity - 100

of 79.2% and a slightly higher sensitivity of 49.4% than the Sokolow-Lyon criterion with well defined ST-T changes. The criterion of $SV3 + R\text{ AVL} > 2.2\text{ mV}$ in men and $> 1.2\text{ mV}$ in women plus positive T wave amplitude in $V1 \geq 0.00\text{ mV}$ in patients ≤ 40 years and $\geq 0.2\text{ mV}$ (in patients > 40 years) was found to have a specificity of 77.1% and a sensitivity of 46.9%. This result could be explained by the fact that T wave amplitude was found to have a low specificity for ECG-LVH. In our study, the Romhilt-Estes criteria did not perform well in the diagnosis of ECG-LVH as they had a specificity of 81.25% and a sensitivity of 40.7%. However, the old Glasgow criteria (appendix 1) had a high specificity of 91.7% for definite LVH (≥ 6 points) and a reasonable sensitivity of 46.8% (table 8.8).

8.14 MULTIPLE LOGISTIC REGRESSION ANALYSIS FOR CALCULATION OF LVH EXPONENT IN PATIENTS WITHOUT MYOCARDIAL INFARCTION

Casale et al (1985) introduced the use of multiple logistic regression analysis for the diagnosis of ECG-LVH. They utilized the BMDP Biomedical computer program suite (Dixon et al 1977) to compare clinical data (age and sex) and ECG parameters with the indexed echo LVM of individual patients. In this approach, the clinical and ECG data are assessed on the basis of yes/no probability to predict the "risk" of ECG-LVH. The value of the risk for ECG-LVH for each individual is represented by R in the following equation:

$$R = 1 / [1 + e^{-(B_0 + B_1.X_1 + \dots + B_k.X_k)}] \quad 8.1$$

where B_0, B_1, \dots, B_k are the logistic coefficients which are estimated from the data that measure levels of k factors (X_1, X_2, \dots, X_k) e.g. age, for each patient. The logistic coefficients are estimated by the method of maximum likelihood.

However, to examine changes of risk with varying levels of one variable at fixed levels of other variables, the previous equation can be linearized to the following equation:

$$\ln [R / (1 - R)] = B_0 + B_1.X_1 + \dots + B_k.X_k \quad 8.2$$

whereby $\ln [R/(1-R)]$ represents the logit of the risk and is the mathematical transformation of the risk. Further, the logit of the risk also allows interpretation of the logistic coefficients B_0, B_1, \dots, B_k .

It follows from equation 8.1 that

$$R = e^a / (1 + e^a)$$

where the exponent a is given by

$$a = B_0 + B_1.X_1 + \dots + B_k.X_k$$

Therefore by using the logistic coefficients B_i for each variable, an exponent is generated for use as a multiple logistic regression equation (MLRE) to predict the risk of ECG-LVH. As a result, multiple logistic regression analysis can provide a quantitative method for assessing

the predictive strength of each variable in a group of inter-related risk factors.

The first MLRE of Casale et al (1985) used for the diagnosis of ECG-LVH, did not take into consideration whether the patients were in sinus rhythm or had atrial fibrillation. However Casale and colleagues in 1987, introduced other two separate MLREs for patients in sinus rhythm and for those who had atrial fibrillation. These were as follows:

Exponent (Sinus rhythm) = $4.558 - 0.092(R\ aVL + SV3)$

$- 0.306T+V1 - 0.212QRSd$

$- 0.278PTFV1 - 0.559sex$

Exponent (Atrial fib) = $5.045 - 0.093(RaVL + SV3)$

$- 0.312T+V1 - 0.325QRSd$

$- 0.602sex$

Partition values of the exponent for the detection of LVH are:

sinus rhythm: $LVH < -1.55$

atrial fibrillation: $LVH < -1.20$

The units of measurement used in the above equations are.

RaVL, SV3 and TV1 in mm (1 mm = 0.1 mV).

QRSd in seconds x 100 (Hundredths of seconds).

PTFV1 in mm x second.

Sex entered as 1.0 for men and 2.0 for women.

The above mentioned multiple logistic regression equations were applied in the present study population of 129 patients for the detection of LVH and assessed. However, these MLREs introduced by Casale et al (1987) did not work satisfactorily in this population. The

specificity was 57.1% for patients in atrial fibrillation and 26.3% for patients in sinus rhythm with sensitivities of 65.2% and 86.9% respectively (table 8.9).

In our population there were 30 patients who had atrial fibrillation (23 with echo LVH and 7 with normal LVH). Another regression equation was therefore developed for these patients as shown below:

$$\begin{aligned} \text{Exponent} = & - 10.968 - 0.028655 \text{ T-aVR} + 0.00505 \text{ SV1} \\ & + 0.008075 (\text{age in months}) \end{aligned}$$

The amplitudes are expressed in microvolts.

The exponent obtained from the present study population using the same approach as Casale did not correlate well with echo LVM in either sex ($r = 0.1439$ for men and $r = 0.3139$ for women). However, despite this poor correlation, an LVH exponent > 2.8 in patients with atrial fibrillation was 100% specific for LVH with a sensitivity of 47.8%. Moreover, when the cut-off point of the LVH exponent was reduced to 0.6, then the specificity reduced to 85% but the sensitivity increased to 78.3%.

There were 99 patients with sinus rhythm in the present study population (61 patients with echo LVH and 38 patients with normal LVM). The regression equation derived for this population is shown below:

$$\begin{aligned} \text{Exponent} = & 1.4985 + 0.00062165(\text{SV3} + \text{RaVL}) \\ & - 0.0089453 \text{ TV6}. \end{aligned}$$

The exponent obtained from this equation for patients in sinus rhythm correlated well with echo LVM

Table 3.9 Specificity and sensitivity of criteria
 derived from regression equations for the
 diagnosis of ECG-LVH.

Criteria	Sensitivity	Specificity

(1) LVMI (Rautaharju et al 1988)		
Men	28.9%	64.3%
Women	38.9%	70.0%
(2) LVMI (Present Study)		
Men	95.6%	42.9%
Women	88.9%	35.0%
(3) Exponent for detection of LVH (Casale et al 1987)		
Sinus rhythm	86.9%	26.3%
Atrial fibrillation	65.2%	57.1%
(4) Exponent for detection of LVH (Present Study)		
Sinus rhythm*	59.0%	92.1%
Atrial fibrillation**	47.8%	100.0%

* LVH diagnosed if the LVH exponent > 0.75

** LVH diagnosed if the LVH exponent > 2.80

in men ($r = 0.4785$) but there was a poor correlation in women ($r = 0.3091$). However, the correlation of the echo LVM and the LVH exponent for both sexes together was good with an r value of 0.4056 ($p < 0.05$) in patients with sinus rhythm. When an LVH exponent > 0.75 was utilized for the diagnosis of ECG-LVH, this criterion was 92.1% specific for LVH with a sensitivity of 59%. Therefore the LVH exponent obtained from our population performed better than the old Glasgow point scoring system for the diagnosis of ECG-LVH both for patients in sinus rhythm and for those who had atrial fibrillation. However these results for exponents only are based on a training set while it is known that invariably test set results are never as good.

8.15 ECG PREDICTED LEFT VENTRICULAR MASS INDEX IN PATIENTS WITHOUT MYOCARDIAL INFARCTION

The estimation of the left ventricular mass index (LVMI) from the scalar ECG was introduced by Rautaharju and his colleagues (1988). They found that the LVMI (g/m^2) substantially identified a larger fraction of subjects with high risk for cardiovascular diseases than the conventional ECG-LVH criteria in a general population. There were separate equations for white and black men, and white and black women. However as negroid patients were not included in the present study, only the following two equations could be used.

$$\begin{aligned} \text{LVMI}(\text{g/m}^2) = & -36.4 + 0.010 \text{ RV5} + 0.020 \text{ SV1} + 0.028 \text{ S}^* \text{ III} \\ & + 0.0182 \text{ T-V6} - 0.148 \text{ T+aVR} + 1.049 \text{ QRSd} \end{aligned}$$

for white and black men, and

$$\text{LVMI}(\text{g}/\text{m}^2) = 88.5 + 0.018 \text{ RV5} + 0.053 \text{ S}^* \text{I} + 0.108 \text{ T+V1} \\ + 1.70 \text{ T-aVF} - 0.094 \text{ T+V6}$$

for white women.

(* = S or Q or QS, whichever is largest, while amplitudes are in absolute values (uV) and QRS duration is in milliseconds).

ECG predicted left ventricular mass index was calculated from the regression equations of Rautaharju et al (1988) for both men and women. Patients with myocardial infarction were excluded as MI reduces the amplitudes of R waves. There was a very poor correlation between the indexed echo-LVH and the ECG predicted LVMI for both men (r value of - 0.1461) and women (r = 0.0528). This poor correlation explained the low specificity in both sexes of ECG predicted LVMI as a criterion for LVH (LVMI > 125 g/m² - both sexes), namely 64.3% and 70% for men and women respectively (table 8.9). The corresponding sensitivities were 28.9% and 38.9%. The limited usefulness of the ECG-predicted LVMI from the equations of Rautaharju et al (1988) was attributed to the fact that these equations were developed in a sample of the general population. Thus it was decided to develop new regression equations for the patients in our own cardiac population. The equations for predicting ECG LVMI were developed by utilizing BMDP Biomedical computer programs (Dixon et al 1977). In this approach, the dependent variables (age, sex and ECG parameters) are

examined individually with the independent variable (echo LVMI). Thereafter, multivariate analysis of the data is carried out by multiple linear regression analysis entering data in a stepwise manner. The sequence of entering variables in the regression equation is determined by conditional correlation of echo LVMI and the preceding variables currently in the equation. The results were as follows:

$$\begin{aligned} \text{LVMI (men)} = & 99.6615 + 0.03358 \text{ SV5} + 0.10121 \text{ T+V1} \\ & - .4231 \text{ T-V1} - 0.12692 \text{ T-V6} \end{aligned}$$

$$\begin{aligned} \text{LVMI (Women)} = & - 115.1371 - 0.04284 \text{ RIII} - 0.02134 \text{ SV3} \\ & + 0.4927 \text{ ST V1} + 3.1725. \end{aligned}$$

The ECG predicted LVMI obtained from these regression equations correlated well with the indexed echo-LVH in both men ($r = 0.5546$) and women ($r = 0.7319$). Unfortunately the ECG-predicted LVMI derived from our regression equations did not perform satisfactorily for the diagnosis of ECG-LVH as it had a specificity of 42.9% and 35% although a sensitivity of 95.6% and 88.9% for men and women respectively.

8.16 EVALUATION OF ECG-LVH CRITERIA IN THE ABSENCE OF MI

The main limitations of ECG-LVH criteria are (i) criteria which are highly specific, have a low sensitivity (ii) highly sensitive criteria have a low specificity and (iii) there is no general agreement on the specificity and sensitivity of any single criterion e.g. the reported specificity of $\text{SV1} + \text{RV5} \geq 3.5 \text{ mV}$

ranges from 25% to 100% with a sensitivity from 21% to 58% (table 3.10).

The reasons for the latter have not previously been studied in detail, with due consideration of all the existing criteria. However, the factors which may be responsible can be divided into two groups:

(I) Inherent limitations of the ECG parameters utilized. These include mainly the PTFV1 and ST-T abnormalities both of which are affected by coronary artery disease, valvular heart disease, pharmacological agents and LV dysfunction, in the absence of LVH.

(II) Multiple factors which affect the QRS voltages including pulmonary oedema and chronic obstructive airways disease.

Therefore it would not be surprising to observe findings in our study which are in agreement with those of others, while some results contradict the conclusions of other researchers, in all probability due to the varying test populations.

8.16.1 Assessment of the non-voltage criteria

Murphy et al (1984) reported that an abnormal PTFV1 performed as efficiently as the voltage criteria in the diagnosis of ECG-LVH. However, Heikkila et al (1973) showed that an abnormal PTFV1 occurs in acute MI without LVH. The present study has also demonstrated that a high abnormal PTFV1 occurs in acute MI in patients who have a low ejection fraction (chapter 3). Recently, Recke et al (1989) have shown that a high PTFV1 was

Table 8.10 The reported specificities and sensitivities of the Sokolow-Lyon
 criteria for LVH i.e. SV1 + RV5 or RV6 \geq 3.5 mV.

Author	Sensitivity	Specificity	Material studied
Romhilt & Estes (1969)	56.3%	25%	PM
Reichek &	21%	95%	PM
Devereux (1981)	53%	86%	Echo
Devereux et al (1984)	22%	93%	Echo
Woythaler et al (1983)	54%	86%	PM & Echo
Casale et al (1985)	33%	94%	Echo/1st series
	20%	93%	Echo/2nd series
Casale et al (1987)	22%	100%	Echo
Kimura et al (1987)	56.6%	76%	PM
Present study	58.0%	64.4%	Echo

associated with LV dysfunction in patients with aortic stenosis. In our study, PTFV1 correlated poorly with the echo LVM ($r = 0.159$) and when an abnormal value ≥ 4.0 mV.msec was used to diagnose LVH, the specificity of the criterion was 81.3% with a sensitivity of 24.8%. Therefore, the criterion of abnormal PTFV1 ≥ 4.0 mV.msec used in isolation is of limited value in the diagnosis of ECG-LVH, contrary to the findings of Murphy et al (1984). Note however that the study of Murphy et al was based on post mortem findings where more severe abnormal pathology may have been present.

Non-specific ST-T changes in leads V5 and/or V6 correlated poorly with left ventricular mass (r values of 0.0934 and 0.0111 respectively). Moreover when the criterion of non-specific ST-T depression was utilized alone for the diagnosis of ECG-LVH, it had a specificity of 25% with sensitivity of 42%. Therefore non-specific ST-T depression in leads V5 and/or V6 has a low specificity for the diagnosis of LVH in a sample of a cardiac population.

ST-T changes that score three points in the Glasgow criteria are defined as a flat or downward sloping ST segment with the J point < -20 uV in leads I, V5 or V6 in the absence of digitalis intake. The T wave inversion also needs to be significant with an amplitude < -200 uV to fulfil the above mentioned criteria provided that it is not associated with pathological Q waves in the same leads. Evaluation of ST-T depression in leads V5, V6 with

T wave inversion < -200 μ V and ST depression < -50 μ V and a slope $< 0^\circ$ used as a single criterion for the diagnosis of LVH resulted in a specificity of 93.7% and a sensitivity of 19.8% compared to a specificity of 89.9% and sensitivity of 35.8% when the ST depression was < -20 μ V and the slope $< -5^\circ$. These results suggest that well defined ST-T changes with computerized measurements are more specific for LVH than non-specific ST-T changes.

Indexed echocardiographic left ventricular masses correlated well with the positive amplitude of the T wave in lead V1 ($r = 0.3708$) but when the criterion of T V1 > 0.00 mV (age < 40 years) and > 0.20 mV (age ≥ 40 years) was assessed as a single criterion, it had a specificity of 54.2% and a sensitivity of 58% for LVH (table 8.7). These results suggest that despite a good correlation between the positive amplitude of the T wave in lead V1 and echo LVH, the T wave voltage criteria included in the Cornell criteria (Casale et al 1987) are not specific for LVH. Moreover, when the amplitude of the T wave was added to the Cornell voltage criteria (Casale 1987), the specificity was still unacceptable (77.1%) with a sensitivity of 46.9% in our study.

The criterion of QRS duration ≥ 0.1 second was only 68.7% specific for LVH with a sensitivity of 44.4% despite a good correlation ($r = 0.3167$) between the QRS duration and the echo LVM. No patient in this study had LBBB, RBBB or intraventricular conduction defect. Left axis deviation $< -30^\circ$ was highly specific (96.4%) for LVH

but with a sensitivity of 9.1% in this cardiac population in which myocardial infarction was excluded (table 8.7). The intrinsicoid deflection ≥ 0.05 second in leads V5/V6 was 97.9% specific with 9.9% sensitivity (table 8.7). The low sensitivity of the left axis deviation and prolonged intrinsicoid deflection does not preclude their use as supporting criteria for the diagnosis of ECG-LVH, because their high specificity allows strengthening of the diagnosis of LVH in the point scoring system.

8.16.2 Specificity and sensitivity of the voltage and the combined criteria

The criterion of SV1 or SV2 > 3.0 mV was 97.9% specific with a sensitivity of 14.8%. A high specificity (95.8%) was observed with the criterion of RV5 or RV6 ≥ 3.0 mV, with 13.6% sensitivity.

The amplitude of the S wave in lead V3 correlated well with the echo LVM in men ($r = 0.3177$) but not in women ($r = 0.0338$). The exact reason for the paradoxical correlation of SV3 with the echo LVM in each sex in this study is not known but it could be due to a larger amount of precordial fat in females. However the amplitude of the R wave in lead aVL correlated with the echo LVM in exactly the opposite manner to that of SV3. There was a poor correlation ($r = 0.118$) between the amplitude of the R wave in lead aVL in men, while the correlation was good in women ($r = 0.4833$). Horton et al (1977) found that the amplitude of the R wave in lead aVL had poorer correlation than the precordial voltages with left

ventricular masses estimated by echocardiography in contradiction to the findings of Casale et al (1987). Despite the poor correlation of R aVL with LVM in men, the criteria of R in lead I and/or R in lead aVL > 2.0 mV had a specificity of 97.9% but a sensitivity of only 6.2%.

The above mentioned results indicate that isolated voltage criteria in single limb or precordial leads are useful for the diagnosis of ECG LVH only when a high cut-off point is used with consequent high specificity and low sensitivity, in agreement with the findings of Allenstein and Mori (1960).

The sum of SV3 and R aVL correlated well with the echo LVM both in men ($r = 0.3548$) and in women ($r = 0.2696$) and thus the combination of these two voltages cancelled the paradoxical correlation of each with LVM in different sexes. The specificity of the criterion SV3 + R aVL > 3.5 mV in men and > 2.5 mV in women (Casale et al 1985) was almost 90% (89.6%) with a sensitivity of 33.3%. However, the Cornell criteria (Casale et al 1987) with or without T wave amplitude in lead V1 did not perform well for the diagnosis of ECG-LVH because of an undesirable low specificity and sensitivity (tables 8.6 and 8.8). These results indicate that the SV3 + R aVL voltage criterion is useful for the diagnosis of ECG-LVH and may perform better if a higher cut-off point is used.

Romhilt and Estes (1968) introduced a point scoring system for the diagnosis of ECG-LVH (table 8.2) and

concluded that the system was 97% specific for LVH with a sensitivity of 54% (table 8.3). Casale et al (1985) reported different specificities for the Romhilt-Estes point scoring system according to whether postmortem or echo data were utilized (table 8.11). The specificity dropped from 95% with postmortem data to 83% on echo data. This illustrates that the specificity of any given criterion could be different when it is assessed against different gold standards even by the same observers. However, this message was not addressed by these authors. Woythaler et al (1983) also reported that the Romhilt-Estes point scoring system was not specific for the diagnosis of ECG-LVH with a specificity of 86% and a sensitivity of 54% (table 8.11). In our study, Romhilt-Estes system was only 81.2% specific with 40.7% sensitivity. An abnormal PTFV1 scores three points in this scoring system and as mentioned earlier in this chapter, PTFV1 is widely affected by diseases other than LVH. This may contribute to false positive results thereby reducing the specificity of the Romhilt-Estes scoring system.

The original Glasgow point scoring system for the diagnosis of ECG-LVH is basically derived from the Romhilt-Estes point scoring system but with modifications which are mentioned in detail in appendix 1. The main modifications are, (1) addition of the voltage criteria of $SV1 + RV5 \geq 3.5$ mV and Lewis index to the Romhilt-Estes voltage criteria, and (2) all voltage

Table 8.11 The reported specificities and sensitivities of Romhilt-Estes point scoring system in the diagnosis of ECG-LVH in the literature.

Author	Sensitivity	Specificity	Material studied
Romhilt Estes (1968)	54%	97%	PM
Reichek Devereux (1981)	50%	95%	PM
Devereux et al (1984)	50%	97%	Echo
≥ 4 points	48%	85%	Echo
≥ 5 points	34%	98%	
Woythaler et al (1983)	54%	86%	Echo
Casale et al (1985)			
≥ 4 points	33%	94% (1st series)	Echo
	31%	83% (2nd series)	Echo
≥ 5 points	19%	96%	
Present study	40.7%	81.2%	Echo

criteria are adjusted for age and sex dependent upper limits of normality. The specificities for possible LVH (≥ 4 points), probable LVH (≥ 5 points) and definite LVH (≥ 6 points) according to the old Glasgow criteria were 77.7%, 88.8% and 91.1% respectively. The corresponding sensitivities were 59.9%, 52.3% and 48.8%. These results clearly reveal that the specificity of the old Glasgow point scoring system for the diagnosis of possible LVH is unacceptable and therefore needs further improvement. Before discussing this further, other aspects of ECG-LVH diagnosis are considered.

8.17 ASSESSMENT OF CONCENTRIC AND ECCENTRIC LVH
WITHOUT MYOCARDIAL INFARCTION FROM THE SCALAR
ECG

8.17.1 ECG parameters used in differentiation of
concentric and eccentric LVH

A study of the ECG parameters that might discriminate between the concentric and eccentric types of LVH was made as follows:

- (I) A comparison was made of the sensitivity of the existing ECG-LVH voltage criteria in concentric and eccentric LVH.
- (II) The incidence of ST-segment depression and T wave inversion in leads V5 and V6, as well as of ST-T segment elevation and tall upright T waves in the same leads was studied with respect to the different types of LVH.
- (III) The prediction of indexed left ventricular volumes

from ECG parameters was assessed for the differentiation of concentric and eccentric types of left ventricular hypertrophy.

(IV) A multiple logistic regression equation was derived to differentiate between concentric and eccentric LVH. For this purpose the Biomedical data analysis program (BMDP) was used in which a two sample t-test is carried out among the patients with left ventricular hypertrophy to find the parameters that exhibit significant differences between the two groups of left ventricular hypertrophy. The following ECG parameters were utilized.

- (1) Age and sex.
- (2) R wave amplitudes in leads I, II, III, aVF, aVL, V4, V5 and V6.
- (3) S wave amplitudes in leads I, II, III, aVL, aVF, V1, V2, V3 and V5.
- (4) ST segment amplitude in leads I, aVL, V5, V6
- (5) Amplitude of P+,P- wave in lead V1.
- (6) Overall duration of the P wave.
- (7) P terminal force in lead V1.
- (8) Intrinsicoid deflection in leads V5 and V6.
- (9) Positive T wave amplitude in leads I, aVL, aVR, aVF, V1, V5 and V6.
- (10) Negative T wave amplitude in leads I, aVL, aVR, aVF, V1, V5 and V6.
- (11) ST segment slope in leads I, V5 and V6.

- (12) QRS and T wave axes
- (13) Duration of the QRS complex.
- (14) QT interval.
- (15) QRS vector.
- (16) ST-T amplitude in leads V5, V6.
- (17) SV1 + RV5
- (18) (RI + SIII) - (SI + RIII)
- (19) R aVL + SV3

8.17.2 Sensitivity of voltage criteria for concentric and eccentric LVH

In view of the fact that increased LV cavity size causes potentiation of the QRS voltages (Brody 1956, Rudy 1987), it might be expected that ECG-LVH voltage criteria would be encountered more frequently in eccentric LVH (LVH and LV dilatation) than in concentric LVH (LVH with normal or reduced LV volume). Therefore it was decided to study the sensitivity of ECG-LVH voltage criteria in the patients with concentric and eccentric types of LVH. The ECG-LVH voltage criteria analysed included:

- (1) SV1 + RV5 or RV6 \geq 3.5 mV
- (2) SV3 + R aVL $>$ 2.8 mV in men and $>$ 2.0 mV in women
(Cornell Voltage criteria)
- (3) SV1 or SV2 \geq 3.0 mV
- (4) RV5 or RV6 \geq 3.0 mV
- (5) R I or R aVL \geq 2.0 mV

A comparison of the sensitivity of the voltage criteria in concentric and eccentric LVH is shown in table 8.12. The Cornell voltage criteria had 51%

sensitivity in both types of LVH, while the sensitivity of the Sokolow-Lyon precordial voltage criterion ($SV_1 + RV_5/RV_6 \geq 3.5$ mV) was 55% in concentric LVH compared to 39% in eccentric LVH. Furthermore, the limb lead voltage criterion of R_I or $R_{aVL} \geq 2.0$ mV was more sensitive in concentric LVH (9%) than in eccentric LVH (2%). However, the criterion of $SV_1/SV_2 \geq 3.0$ mV has a sensitivity of 13% and 14% for concentric and eccentric types of LVH respectively. A similar trend was observed for the criterion of $SV_1/SV_2 \geq 3.0$ mV, as it had a sensitivity of 14% and 13% for eccentric and concentric LVH respectively. These results suggest that -

- (i) there is no difference between the concentric and eccentric types of LVH with regard to the sensitivity of an ECG criterion that combines both limb and precordial voltages such as the Cornell voltage criteria;
- (ii) patients with concentric LVH had isolated limb lead ($R_I/R_{aVL} \geq 2.0$ mV) and Sokolow-Lyon precordial voltage criteria more frequently positive than those with eccentric LVH;
- (iii) isolated precordial voltage criteria such as $SV_1/SV_2 > 3.0$ mV, and $RV_5/RV_6 > 3.0$ mV are more sensitive in eccentric than in concentric LVH; and
- (iv) therefore there is a trend that concentric LVH causes more potentiation of the R waves in leads I and/or aVL than eccentric LVH. On the other hand higher voltages in SV_1/SV_2 and RV_5/RV_6 resulted from eccentric rather than concentric LVH. However, although an extremely high

Table 8.12 Sensitivity of the voltage criteria for
concentric and eccentric types of left
ventricular hypertrophy.

Criteria	Sensitivity in Concentric LVH	Sensitivity in Eccentric LVH

SV3 + R aVL		
> 2.8 mV (men)	51%	51%
> 2.0 mV (women)		
SV1 or RV5 or RV6 \geq		
3.5 mV	55%	39%
SV1 or SV2 \geq 3.0 mV	13%	14%
RV5 or RV6 \geq 3.0 mV	11%	14%
R I or R aVL \geq 2.0 mV	9%	2%

R voltage in leads I and/or aVL provides 90% probability that the LVH is of concentric type, the occurrence of such voltage criteria in smaller numbers of patients with eccentric LVH, makes this criterion of limited value in the differentiation of these two types of LVH in individual patients.

8.17.3 ST-T abnormalities in concentric and eccentric LVH

36 patients with definite ECG-LVH on the Glasgow criteria and documented echo LVH, were studied with respect to the pattern of the ST segment and T wave changes. Echocardiographically they included two groups, namely -

(i) 19/36 (52%) of the patients had concentric LVH, and an indexed LVM of $196.0 \pm 64.4 \text{ g/m}^2$ (range 116 - 339 g/m^2) and mean indexed LVV of $52.63 \pm 19.22 \text{ ml/m}^2$ (range 26 - 86),

(ii) 17/36 (48%) of the patients had eccentric LVH, and an indexed LVM of $220.8 \pm 67.8 \text{ g/m}^2$ (range 139 - 344) as well as dilated left ventricles (mean indexed LVV of $142.5 \pm 42.9 \text{ ml/m}^2$ and range of 91 - 238 ml/m^2).

There were 8 patients who had atrial fibrillation (4 patients with concentric and 4 patients with eccentric LVH). All of these 8 patients were on digoxin. The frequency of ST-segment depression and T wave inversion are shown in table 8.13. Asymmetric ST-T changes were observed more frequently in eccentric LVH (volume overload) than in concentric LVH (pressure overload).

Therefore the electrocardiographic concept of systolic and diastolic overload based on the pattern of ST-T changes in anterolateral leads does not seem able to differentiate between concentric and eccentric LVH.

8.17.4 Prediction of left ventricular volumes from the scalar ECG

The biomedical data analysis program was utilized to develop regression equations to predict indexed left ventricular volumes (LVVI) from the scalar ECG. This was done by correlating the ECG parameters with echo LVVI as described earlier in two populations of patients namely (i) the total population of patients including those with normal echo LVM and those with echo LVH, (ii) only those patients with echo LVH

In the total population (129 patients), the only ECG parameter which correlated well with the indexed LVV was QRS duration ($r = 0.2506$, $p < 0.0038$) and therefore the following regression equation was developed:

$$\text{ECG LVVI (ml/m}^2\text{)} = -17.7247 + 1.06482 \text{ QRSd(msec)}$$

However, in the population with echo LVH only (84 patients) the following ECG parameters correlated with echo LVVI namely, ST segment in lead V6 ($r = 0.3001$, $p = 0.048$), QRS duration in lead V5 ($r = 0.1861$, $p = 0.0462$). The amplitude of S in lead V1 was negatively related to the left ventricular volume ($r = 0.113$) but this negative correlation was not significant ($p = 0.3013$). Therefore the following regression equation was developed to predict indexed LVV:

Table 8.13 Pattern of ST-segment and T wave in
patients with ECG-LVH proved by echo-
cardiography and definite ECG-LVH. All
the patients with atrial fibrillation were
on digoxin.

Types of LV	No ST-T changes	Asymmetric ST-T inversion	Symmetric ST-T depression

Concentric			
n = 19	3	12	4
Eccentric			
n = 17	2	15	2

$$\text{ECG LVVI}(\text{ml/m}^2) = 58.44646 - 0.0187(\text{SV1}) + 0.30825(\text{STV6}) \\ + 2.33063(\text{QRSd V5})$$

where amplitudes are in uV and QRS duration is in msec.

However, unfortunately, the ECG predicted LVVI derived from the above regression equations did not perform satisfactorily for the diagnosis of eccentric LVH (LVH with dilated LV). The specificity of the ECG predicted LVVI for the diagnosis of eccentric LVH among the total population was 69.3% in comparison to 44.2% in the smaller echo LVH population using the two separate equations. The corresponding sensitivities were 48.8% and 73.2% respectively. These results indicated that ECG predicted LVVI is of limited value in the diagnosis of eccentric LVH and hence in differentiating eccentric from concentric LVH.

8.17.5 Multiple logistic regression equation in differentiation of concentric and eccentric types of LVH

84 patients with echo LVH were included in this study and they consisted of 43 patients with concentric LVH and 41 patients with eccentric LVH. The analysis of their clinical data (age and sex), ECG parameters (mentioned earlier under section 8.17.1) and echo parameters such as indexed LVM and LVV, was made by the Biomedical Computer Program (BMPD). The analysis revealed that the parameters which were significantly different among the concentric and eccentric types of LVH were the sex ($p < 0.05$) and negative T wave amplitude in lead aVL ($p < 0.05$). A

multiple logistic regression equation was developed to try to differentiate between those two types of LVH as follows:

$$\text{Exponent} = 0.27506 - 0.45471 \text{ Sex} + 0.003234 \text{ TaVL}$$

where sex is entered as 1 for men and 2 for women, and the amplitudes are in microvolts (uV).

An exponent > -0.5 (less negative than -0.5) was found in 8 and 17 patients with concentric and eccentric types of LVH respectively. Therefore the corresponding sensitivity for this criterion was 18% for concentric LVH compared to 41% for eccentric LVH. These results indicate that exponents derived from multiple logistic regression in a sample of LVH cannot separate concentric from eccentric types of LVH.

8.17.6 Is it possible to differentiate between concentric and eccentric LVH from the scalar ECG in individual patients?

Cabrera and Monroy (1952) suggested that pressure and volume overload types of LVH could be differentiated by surface ECG from the pattern of the ST-T changes in leads V5 and V6. They reported that asymmetric ST-segment depression and T wave inversion in leads V5 and V6 are characteristic of pressure overload such as in hypertension and aortic stenosis, while the volume overload situations such as aortic and mitral regurgitation produce upright T waves and taller R waves

in the same leads. However Devereux and Reichek (1982) observed that asymmetric ST depression and T wave inversion of LV strain was more frequently found in patients who had increased left ventricular internal dimensions on M-mode echocardiograms than those with normal LVID at end diastole. Moreover they also showed that ECG LV strain did not correlate with the echocardiographic LV wall thickness. In our study, asymmetric ST depression and T wave inversion was also observed more frequently in eccentric LVH (88%) than with concentric LVH (84%) (figures 8.2 and 8.3) which indicates that the ECG pattern of LV strain is not helpful in differentiating between these two types of LVH (table 8.13). Furthermore, significant LVH occurred without ST-depression and T wave inversion, while symmetrical T wave inversion was also observed in LVH but less frequently than asymmetric T wave inversion. In addition, the indexed left ventricular volumes estimated from the scalar electrocardiograms did not reliably predict the actual volumes. Therefore attempts to differentiate between concentric and eccentric types of LVH appear to be unsuccessful.

It is difficult to assess the effects of LV volume on the QRS voltages in two different groups of patients who have different echo LVMS with individual variations due to the effects of thoracic volume conductors. The sensitivity of the ECG-LVH voltage criteria in the limb leads was higher for concentric than eccentric LVH, in

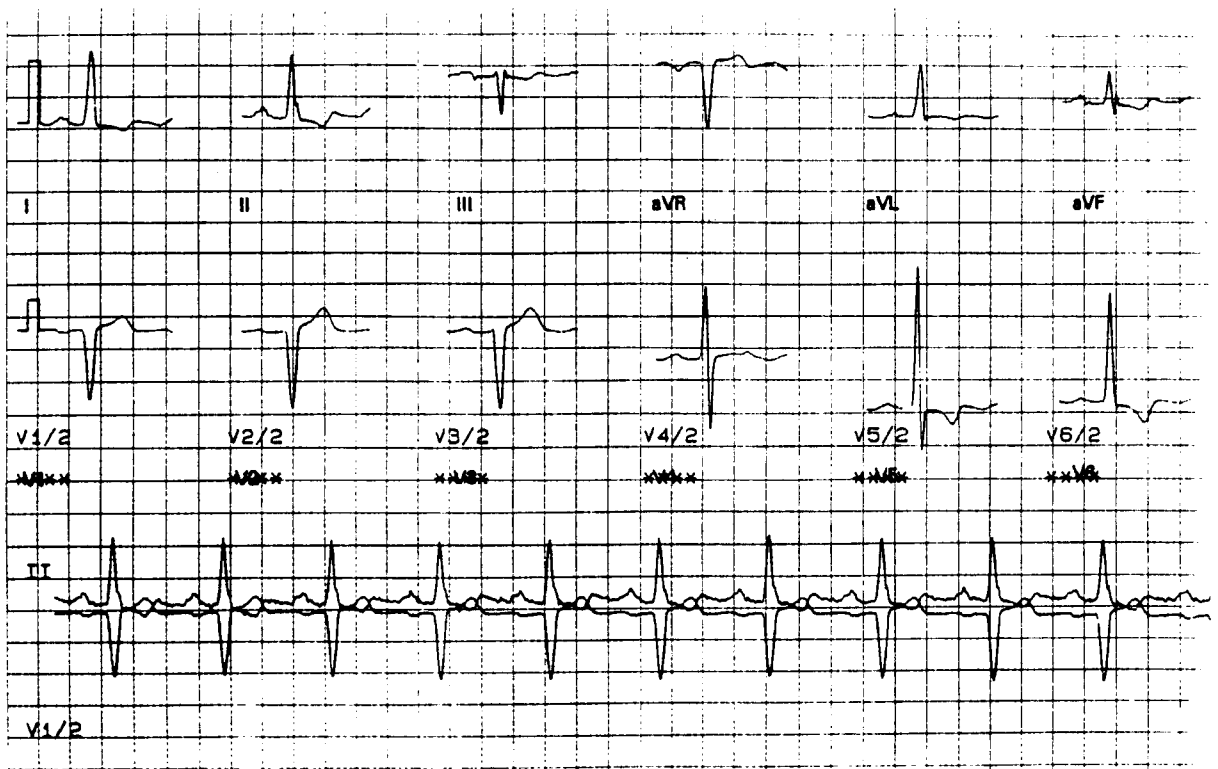


Figure 8.2

Electrocardiogram of a 56 year old man with pure aortic stenosis with normal coronary arteries who underwent aortic valve replacement. The ECG shows left ventricular strain in addition to the voltage criteria of left ventricular hypertrophy both in the limb and the precordial leads. The echo left ventricular mass was 246 g/m² (Penn convention).

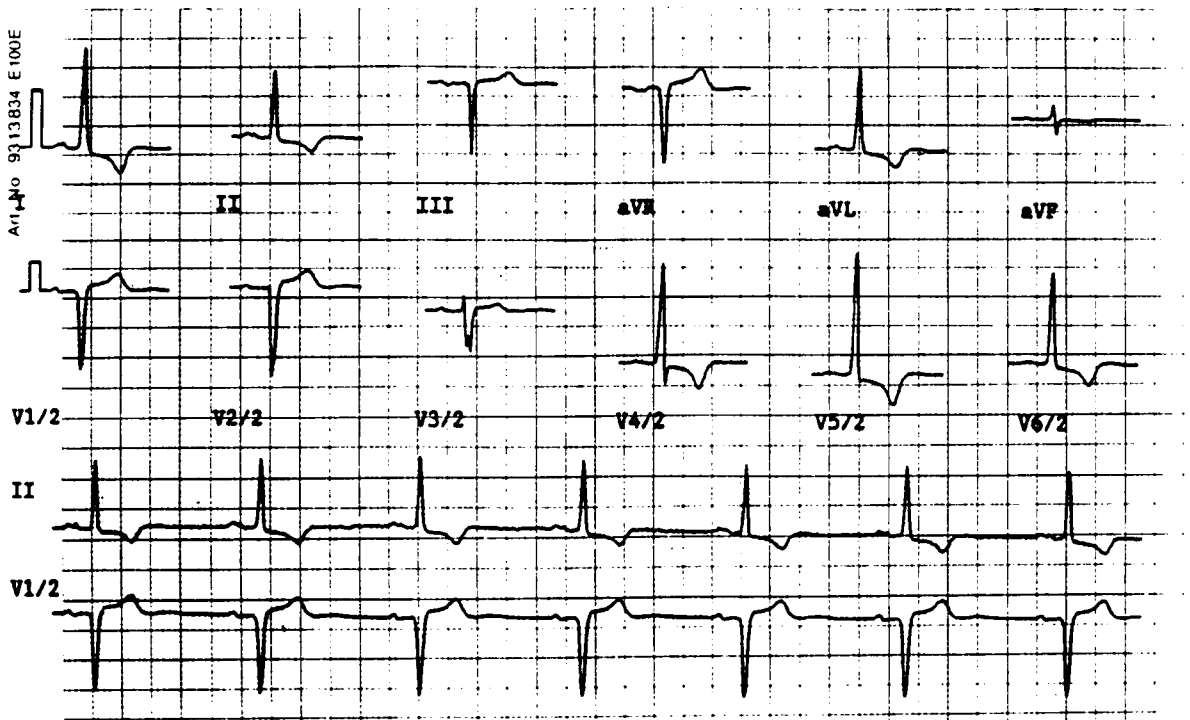


Figure 8.3

Electrocardiogram of a 67 year old man with pure aortic regurgitation and normal coronary arteries who underwent aortic valve replacement. The ECG shows left ventricular strain in addition to the voltage criteria of left ventricular hypertrophy both in the limb and precordial leads. The echo left ventricular mass was 220 g/m^2 (Penn convention).

contrast to the isolated precordial voltage criteria of $SV1/SV2 \geq 3.0$ mV or $RV5/RV6 \geq 3.0$ mV which had a higher sensitivity in eccentric than concentric LVH. However despite these differences, it is difficult to differentiate between these two types of LVH on the basis of ECG-LVH voltage criteria in individual patients.

The sex and negative T amplitude in lead aVL were significantly different between the concentric and eccentric types of LVH ($p < 0.05$) and therefore they were utilized to develop a multiple logistic regression equation for detection of an exponent that can be used as a discriminant between concentric and eccentric types of LVH. An exponent > -0.5 was found in 17/43 (40%) and 8/41 (23%) of the patients with eccentric and concentric LVH respectively which resulted in a specificity of 81.4%. This finding also indicates that this approach was unsuccessful in differentiating these two types of LVH in individual patients.

8.18 IMPROVEMENT OF THE POINT SCORING SYSTEM FOR ECG-LVH DIAGNOSIS

Obviously a good criterion is one which is highly specific while retaining a high sensitivity and which is reproducible by other authors in a sample of both a general and a cardiac population. This can only be evaluated by a critical appraisal of the existing criteria against left ventricular masses. However the ECG parameters and criteria utilized for the diagnosis of LVH are numerous, which makes such evaluation by

manual measurements both tedious and unreliable, e.g. the ECG data derived from a study that is composed of 129 patients would consist of more than 7000 measurements. Therefore, only computer analysis of such data would be able to produce a comprehensive view of the ECG-LVH criteria. The computerized measurements of the ECG waveforms were mainly used in two ways to test the Glasgow ECG-LVH criteria:

- (1) By omission of criteria which did not correlate well or were not highly specific for LVH such as the P terminal force in lead V1.
- (2) By addition of other voltage criteria to the point scoring system such as those of Casale et al 1985, 1987.

The P terminal force in lead V1 neither correlated well ($r=0.159$) nor was highly specific for LVH (specificity = 81.3%). When it was omitted from the old Glasgow criteria (≥ 6 points), the specificity increased marginally to 93.3% but the sensitivity decreased from 46.8% to 32.1%. Therefore it appears that if this criterion is not included in the point scoring system, the specificity would increase moderately but there would be a major reduction in the sensitivity. This indicates that keeping it in the scoring system would be useful but perhaps a lesser weight is needed for scoring. However, when another definition of the P terminal force in lead V1 was used, i.e. the negative deflection of the P wave < -100 μ V PLUS its duration ≥ 40 ms, this criterion alone

became highly specific (91.1%) but with a sensitivity of 17.9%.

When the Cornell voltage criteria (Casale et al 1987) were added to the old Glasgow criteria, the specificity was unacceptable at levels of 75.5% but there was a higher sensitivity of 61.9% (table 8.8).

Moreover the addition of $SV_3 + R aVL > 3.5$ mV in men and > 2.5 mV in women to the old Glasgow criteria lowered the specificity to 84.4% while it increased the sensitivity (≥ 6 points) to 55.95%.

The results of the specificities and sensitivities of the non-voltage criteria excluding well defined ECG LV strain indicate that these criteria are SUPPORTING criteria rather than essential criteria and thus each one alone should not score more than two points in the point scoring system for the diagnosis of ECG-LVH. Well defined LV strain alone was 39.9% specific and 35.8% sensitive. Therefore LV strain as defined above was the best individual ECG parameter for the diagnosis of LVH and therefore it should score more points than even the age and sex dependent voltage criteria. Thus accordingly it was decided to modify the scoring system as follows:

- (1) Voltages beyond the age and sex dependent upper limits of the criteria included in the old system score 2 points as opposed to 3 originally.

However, as in the old system (appendix 1), one extra point is awarded for additional 0.3 mV over the limit in limb leads and one extra point for

every 0.5 mV over the limit in precordial leads in patients ≥ 17 years old. However, one point is deducted if there are Q waves in the anteroseptal leads together with high precordial voltages.

- (2) LV strain is defined (in leads I, VL, V5, or V6) when ST segment depression is < -20 uV with a slope < -5 degrees or ST segment depression is < -50 uV with a slope < 0 degrees, in association with T wave inversion exceeding -200 uV, or if T is biphasic with $T < 0$ mV and with a terminal overshoot < 0.150 mV. However this definition of LV strain is applied if there are no Q waves in the lateral leads and if the amplitude of the R or R' in these leads exceeds 1.0 mV. In addition the QRS duration must not exceed 120 msec. No account is taken of digitalis in this system. This definition of LV strain would score four points. However, if the above definition of LV strain is associated with low amplitudes of the R or R waves in the lateral leads then it would score only two points. If inferior MI is present in addition with $T-aVF < -0.05$ mV, two points are deducted.
- (3) Any ST-T changes in the lateral leads together with high voltages as in (1) score two points if (2) above is not true.
- (4) The supporting criteria of LVH would score as follows:
- (a) P terminal force in lead V1 ≥ 4.0 mV msec.

scores two points. One point is given for a patient who has atrial fibrillation.

- (b) Left axis deviation superior to -30 degrees (QRS axis between -30 and -120 degrees) scores two points in the patient who has no inferior infarction.
- (c) QRS duration > 0.10 second in leads V5 or V6 scores one point provided that the patient does not have right bundle branch block (RBBB) RBBB + left anterior fascicular block (LAFB), RBBB + left posterior fascicular block, or intraventricular conduction defect (IVCD).
- (d) Intrinsicoid deflection in V5 or V6 > 60 msec. without Q waves in the same leads, scores one point because this criterion has a low sensitivity.

The computer would interpret the scoring system for the electrocardiographic diagnosis of LVH as follows:

- (1) Left ventricular hypertrophy (≥ 6 points).
- (2) Probable LVH (5 points)
- (3) Possible LVH (4 points)
- (4) LVH with secondary ST-T changes is reported when the LVH score includes any of the two versions of LV strain. If the patient is on digoxin, then the report would be "LVH with ST-T changes probably due in part to myocardial ischemia/digitalis effect".
- (5) Possible LVH, probable LVH or LVH without LV

strain but associated with $T < -200$ uV in I, aVF V5, V6 without infarction would be reported as "LVH with ST-T changes likely to be due in part to myocardial ischemia".

The study population was assessed with this new scoring system. The indexed left ventricular masses correlated well with the scores of the new scoring system ($r = 0.3718$). Furthermore, the new system resulted in an improved diagnosis of ECG-LVH compared to the old score (table 8.14). The specificity for a score ≥ 4 points increased from 77.7% on the old score, to 86% on the new scoring system while the sensitivity also increased from 59.5% to 64%. The latest finding was the only situation where an increase in specificity was not associated with reduction in the sensitivity. The specificity for definite LVH (≥ 6 points) increased from 91.1% on the old Glasgow system to 93% according to the new system although use of the new approach resulted in a slight drop of sensitivity from 48.8% (old Glasgow system) to 47%. The new Glasgow scoring system proved to be more accurate than the old system when both were assessed by the Association Index (see Macfarlane and Lawrie 1989) where:

$$\text{Association Index} = \text{Sensitivity} + \text{Specificity} - 100$$

The association index for possible LVH increased from 37.6 to 50 on the new system i.e. by 33%. It can be seen

Table 8.14 Comparison of the specificity and
 sensitivity of the old and new Glasgow
 point scoring system for ECG-LVH diagnosis.

Score points	(ECG-LVH)	Old System	New System

≥ 4 points	Specificity	77.7%	86%
	Sensitivity	59.9%	64%
	Association Index	37.60	50.0
≥ 5 points	Specificity	88.8%	86%
	Sensitivity	52.3%	57%
	Association Index	41.10	43.0
≥ 6 points	Specificity	91.7%	93%
	Sensitivity	46.8%	47%
	Association Index	39.90	40.0

from table 8.14 that the new system is by far the best of all criteria studied. Although 86% may not be excessively high for specificity in this group, there were two patients who might have been described as having LVH by ECG only. Figure 8.4 shows the ECGs of these patients with LV masses of 94 g/m^2 and 104 g/m^2 respectively. Their inclusion as LVH would have produced 93% specificity both for possible LVH (≥ 4 points) and probable LVH (≥ 5 points) and 97% specificity for definite LVH (≥ 6 points) on the new Glasgow point scoring system. Furthermore, the corresponding sensitivities would have been 65%, 58% and 48% for possible, probable and definite LVH respectively.

The 10 patients with decompensated LV i.e. normal LV masses and increased volumes were excluded for reasons indicated earlier. The new Glasgow point scoring system did not report LVH in any of these patients. Therefore inclusion of this group as additional normals (control) would have improved the specificity of the new Glasgow scoring system from 86% to 90% for both possible (≥ 4 points) and probable LVH (≥ 5 points), and from 93% to 95% for definite LVH (≥ 6 points).

The new Glasgow point scoring system was also evaluated in a test series as a part of the second phase of the CSE (Common Standards for Electrocardiography) study (Willems et al 1990). In this study, there were 398 healthy subjects and 72 patients with echo LVH. The new Glasgow scoring system diagnosed ECG-LVH (≥ 4 points)

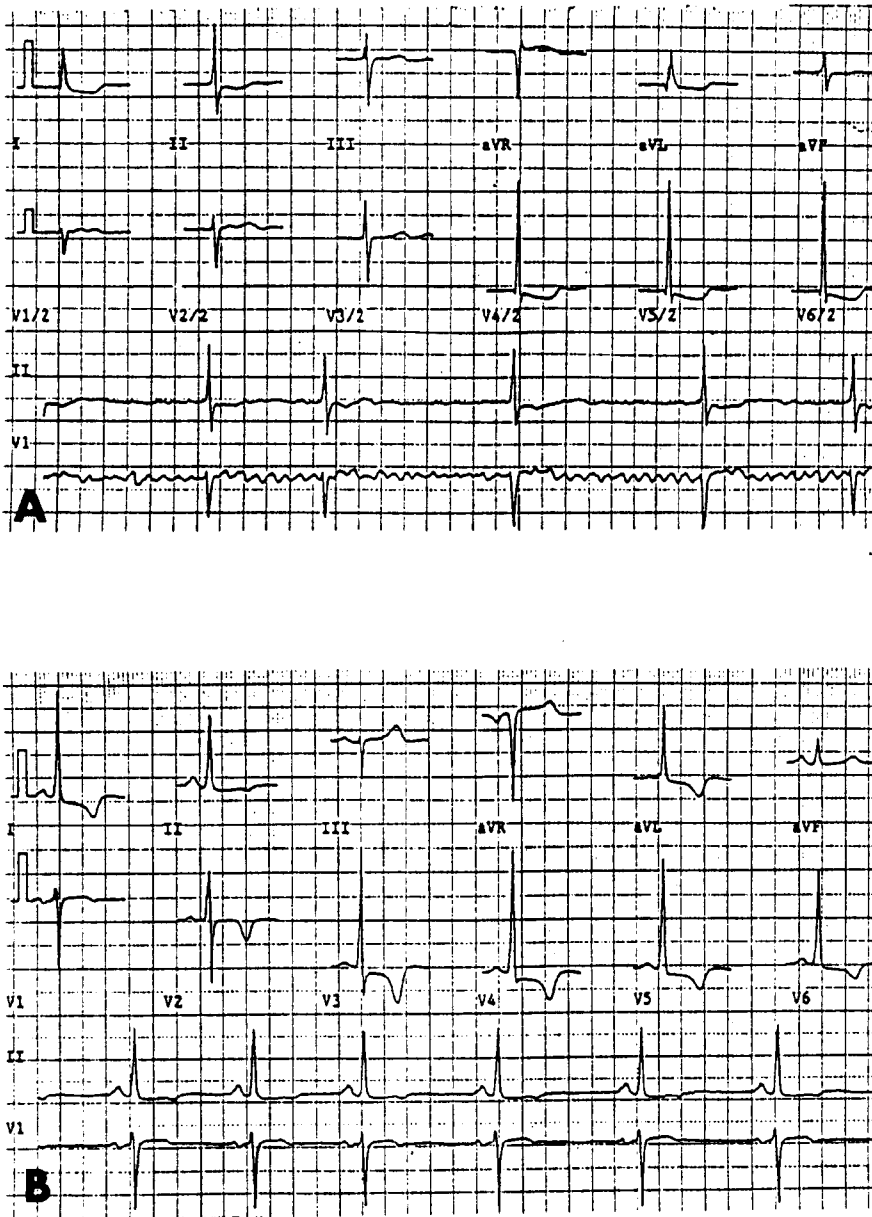


Figure 8.4

ECGs of two patients with normal echo LVMI. The ECG (A) belongs to a 64 year old lady with mitral valve disease and an echo LVMI of 104 g/m^2 while her ECG shows very high precordial voltages and LV strain. The ECG (B) belongs to another lady (48 years old) with hypertension and an echo LVMI of 84 g/m^2 . It shows LV strain and high voltages in the limb leads. In both cases, the heart is classed as normal as $\text{LVMI} < 109 \text{ g/m}^2$.

in 7/398 subjects without LVH and in 47/72 patients with LVH. Therefore the new Glasgow scoring system achieved a high specificity (98.2%) for possible LVH (≥ 4 points) in a test series with a sensitivity of 65% (Association Index = 63). These results indicate that the high specificities and sensitivities achieved with the new Glasgow point scoring system were reproducible in a test population.

8.19 ECG DIAGNOSIS OF LVH IN THE PRESENCE OF
ANTEROSEPTAL Q WAVES

8.19.1 ECG criteria analysed in patients with QS
deflections in V1-V4

The following ECG criteria were analysed in 45 patients who had QS deflections in leads V1-V4:

- (1) Precordial voltage criteria SV_1 (QS V1) + $RV_5/$
 $RV_6 \geq 3.5$ mV.
- (2) Frequency of R I > 1.3 mV and R aVL > 1.1 mV.
- (3) Glasgow criteria for ECG-LVH.

Furthermore, the amplitudes of the deepest SV_1 or SV_2 , tallest RV_5 or RV_6 , and the R waves in leads I and aVL, all in the patients with antero-septal MI without echo-LVH, were compared to the age and sex dependent upper limits of normal (table 8.1).

The above mentioned ECG-LVH criteria were assessed in comparison with echo LVM as the gold standard. This approach has been criticized earlier by Woythaler et al (1983) because MI leads to (i) thin scarred walls with isolated septal hypertrophy, and (ii) alterations of LV

geometry due to wall motion abnormalities that impose limitations on the measurement of LV volumes and all other secondarily derived measurements (Trichholz, Kreuleu, Herman and Gorlin 1976). However, in this thesis, echo LVM was utilized for evaluation of ECG-LVH criteria in the population of patients without MI. Therefore, it was decided to utilize the same approach for the patients with antero-septal MI in order to retain consistency in the methodology for evaluation of ECG-LVH criteria. This is particularly important because specificities and sensitivities of individual ECG-LVH criteria vary considerably with respect to the gold standard adopted for evaluation, as has been mentioned in detail in section 18.6

8.19.2 Data analysis of the patients with antero-septal Q waves

45 patients had pathological Q waves in V1-V2 and sometimes additional precordial leads. They included 26 men, 19 women with a mean age of 56.7 ± 10.5 years and an age range of 38 to 91 years. Identification of the patients with antero-septal myocardial infarcts as a cause of the pathological Q waves in precordial leads, was made from clinical history and cardiac enzymes, with or without Thallium 201 scans or contrast left ventriculograms. Left ventricular mass (LVM) was calculated with the Penn convention, indexed to the body surface area (BSA). It ranged from 58 to 420 g/m^2 with a mean of $177.64 \pm 76.2 \text{ g/m}^2$. Patients were divided into

two echocardiographic groups according to their echo indexed LVM as follows:

(I) Patients with normal indexed LVM, all of whom had antero-septal myocardial infarcts. They included 13 patients (8 men, 5 women, mean age 53.46 ± 8.62 years with an age range of 36 to 66 years). Their echo indexed LVM ranged from 58 to 127 g/m^2 with a mean of $100.69 \pm 21.5 \text{ g/m}^2$. The distribution of the ECG parameters of deepest SV1 or SV2, tallest RV5 or RV6, R I, R aVL, SV1 + RV5/RV6 and the Glasgow score of these patients is shown in table 8.15.

(II) Patients with echo LVH who had pathological Q waves in the antero-septal leads. They included 32 patients (18 men, 14 women, mean age 57.6 ± 11.6 years and an age range of 32-81 years. Their echo indexed LVM ranged from 134 to 420 g/m^2 with a mean of $208.9 \pm 67.6 \text{ g/m}^2$. However, this second group of patients was also subdivided into two other groups according to the presence or absence of antero-septal myocardial infarcts as follows:

(i) Antero-septal MI with echo LVH (figure 8.5).

This group included 20 patients (13 men, 7 women, mean age 59.8 ± 10.2 , age range 44 to 81 years). Their mean indexed echo LVM was $213.8 \pm 73.8 \text{ g/m}^2$ (range 134.0 - 420 g/m^2). The distribution of the ECG parameters of the deepest SV1 or SV2,

Table 8.15 Electrocardiographic and echocardiographic data of patients with anteroapical myocardial infarction without left ventricular hypertrophy.

Pt.	Sex	Age (yrs)	LVM* g/m ²	Q waves	New Glasgow criteria	SV1+RV5/RV6 mV	RI mV	RaVL mV	SV1/SV2 mV	RV5/RV6 mV
CC	F	56	98	V1-V3	N	2.0	1.0	0.8	1.5	0.5
SF	M	43	127	V1-V2	N	2.7	0.4	0.4	2.6+	1.05
JH	F	59	101	V1-V4	N	5.0+	0.7	0.5	2.4+	4.0+
WD	M	50	116	V1-V3	N	2.3	0.4	0.15	1.7	2.0
TM	M	60	127	V1-V4	N	3.2	1.3	1.2	2.5+	1.25
JC	M	38	73	V1-V4	N	1.3	0.7	0.7	1.1	0.4
JO	M	50	130	V1-V3	N	2.45	0.8	0.7	1.9	0.55
RY	M	58	101	V1-V2	N	4.2+	1.5	1.1	4.2+	2.4
WS	M	48	58	V1-V2	N	1.7	0.5	0.7	1.9	0.4
AC	F	40	82	V1-V3	N	1.7	0.7	0.4	1.65	0.6
RB	F	58	102	V1-V3	N	2.1	0.9	0.5	1.4	0.7
HP	F	66	90	V1-V3	N	1.8	0.7	0.3	1.3	0.5
DC	M	64	104	V1-V3	N	1.3	0.8	0.5	0.8	0.5

N Negative Glasgow criteria

* Indexed LVM calculated according to Penn Convention

+ Indicates that the voltage exceeds the age and sex dependent upper limits of normal

tallest RV5 or RV6, SV1 + RV5 or RV6, RI, R aVL, and the new Glasgow criteria are shown in table 8.16.

(ii) Echo LVH without anteroseptal MI. This group included 12 patients (5 men, 7 women, mean age 54.4 ± 13.5 years, age range 32 to 72 years). An example of electrocardiographic pseudo MI with anteroseptal Q waves due to LVH was shown earlier (figure 8.1). Their mean indexed echo LVM was $200.9 \pm 57.6 \text{ g/m}^2$ (range 134 - 350 g/m^2). Anteroseptal myocardial infarction in all these patients was excluded by either Thallium 201 scan and/or contrast ventriculograms. All had aortic valve disease except two (one with systemic hypertension and the other with mitral valve disease). The distribution of the deepest SV1 or SV2, tallest RV5 or RV6, SV1 + RV5 or RV6, mV, R I, R aVL, and the new Glasgow criteria are shown in table 8.17.

The following results were obtained:

- (1) In the patients with anteroseptal MI and a normal indexed LVM, no patient had voltages of R I or R aVL increased beyond the age and sex dependent upper limits of normal values (table 8.14). However, 4/13 (30%) and 1/13 (7%) had abnormally high voltages of SV1/SV2 and RV5/RV6 respectively

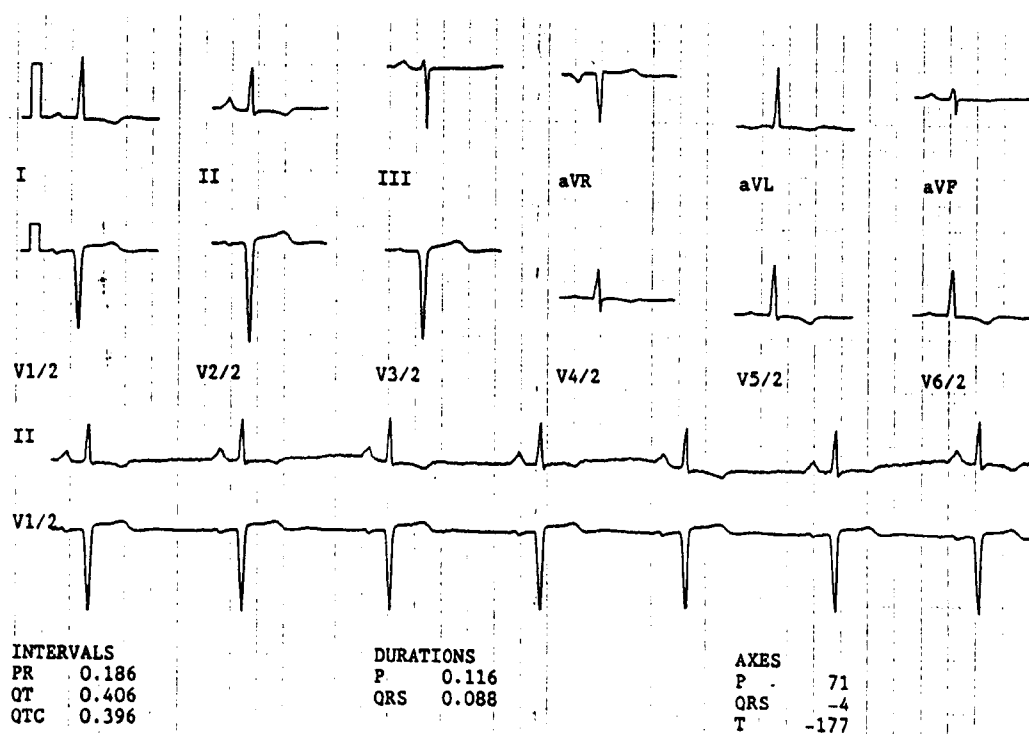


Figure 8.5

ECG of a 57 year old man with an old Q wave antero-septal MI and LVH. His echo LVM was 291 g/m^2 . The ECG meets both limb and precordial voltage criteria of LVH.

Table 8.16 Electrocardiographic and echocardiographic data of the patients with anteroseptal myocardial infarction and left ventricular hypertrophy.

Pt.	Sex	Age (yrs)	LVM* g/m ²	Q waves V1-V4	Glasgow criteria	SV1+RV5/RV6 mV	RI mV	RaVL mV	SV1/SV2 mV	RV5/RV6 mV
CK	F	51	144	V1-V2	-	1.6	0.8	0.8	1.3	0.5
CC	F	78	286	V1-V2	+	3.6	1.3	1.1	2.8	2.2
AT	M	81	259	V1-V4	-	2.3	0.7	0.7	1.9	0.8
JF	M	51	181	V1-V4	-	1.3	0.3	0.7	2.1	0.25
WC	M	57	164	V1-V4	-	2.3	0.6	0.2	2.0	1.3
SM	M	71	134	V1-V2	+	3.7	1.0	0.8	1.8	2.1
AR	F	68	199	V1-V3	+	4.0	2.0	1.2	2.6	1.7
JR	F	68	420	V1-V3	+	2.8	1.9	1.5	3.4	0.6
AS	M	50	143	V1-V2	+(Prob.)	1.9	1.0	1.4	2.3	0.3
JM	M	66	289	V1-V6	-	1.5	0.3	0.2	1.9	0.5
JD	M	66	163	V1-V2	-	2.1	0.4	0.2	2.3	1.1
JB	M	44	134	V1-V2	+	3.4	0.9	0.5	2.1	1.9
AC	M	57	291	V1-V2	+	3.6	2.2	2.6	2.35	1.95
DH	M	52	188	V1-V3	+(poss.)	3.3	2.1	1.9	2.2	1.0
CH	M	54	159	V1-V3	+	2.5	1.0	1.0	1.7	1.5
CHO	M	54	251	V1-V3	-	2.9	0.9	0.5	1.45	1.8
AL	M	54	186	V1-V3	+(prob.)	2.0	0.5	0.2	1.2	2.0
CD	F	57	171	V1-V2	-	2.8	1.2	1.0	1.5	1.5
AL	M	66	221	V1-V2	-	3.7	1.4	1.1	1.5	2.8
JG	M	48	291	V1-V2	-	4.6	1.1	1.0	3.6	1.8

* Indexed LVM calculated on Penn Convention

(table 8.15).

- (2) The total numbers of patients who had positive Glasgow criteria (new), $SV_1 + RV_5/RV_6 \geq 3.5$ mV (Sokolow-Lyon 1949), $R_I > 1.3$ mV (Manning and Smiley 1964), and $R_{aVL} > 1.1$ mV (Sokolow-Lyon 1949) in the three groups of patients are shown in table 8.18.

The Glasgow score (new scoring system) was normal in all the patients with antero-septal MI without echo LVH while the Sokolow-Lyon criterion of $SV_1 + RV_5/RV_6 \geq 3.5$ mV was positive in 2/13 (15%), $R_I \geq 1.3$ mV in 1/13 (7%), and $R_{aVL} \geq 1.1$ mV in 1/13 (7%) of the patients who had a normal indexed left ventricular mass but with Q-wave antero-septal MI. Figure 8.6 shows an ECG of a 58 year old man with acute antero-septal MI and normal echo LVM (101 g/m^2) and false positive Sokolow-Lyon precordial voltage criterion $SV_1 + RV_5/RV_6 \geq 3.5$ mV. However the new Glasgow score was positive in 10/20 (50%) and 9/12 (75%) of the patients with antero-septal MI with echo LVH and in those who had echo LVH without MI respectively. The Sokolow-Lyon precordial voltage criterion was positive in 6/20 (33%) patients with echo LVH and Q wave antero-septal MI, while 8/12 (66%) of the patients with echo LVH with Q waves in antero-septal leads but who had no antero-septal MI, had positive Sokolow-Lyon precordial voltage criteria. The

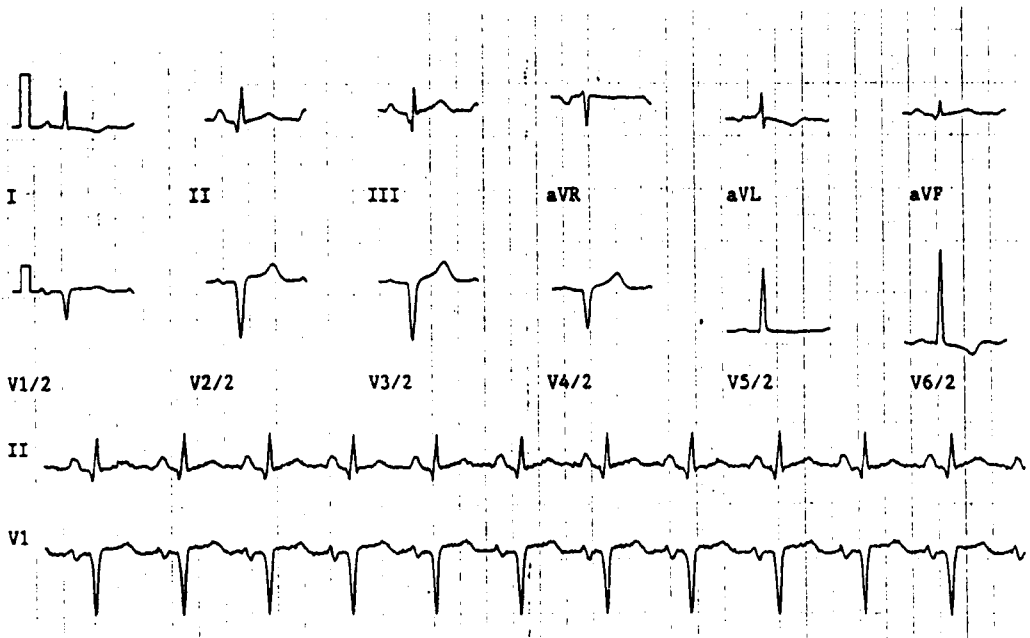


Figure 8.6

ECG of a 58 year old man with acute Q wave antero-septal MI and normal echo LVM (101 g/m^2). The ECG shows false positive Sokolow-Lyon precordial voltage criterion ($\text{SV}_1 + \text{RV}_5/\text{RV}_6 \geq 3.5 \text{ mV}$).

Table 8.17 Electrocardiographic and echocardiographic data of patients with left ventricular hypertrophy without anteroseptal myocardial infarction.

Pt.	Sex	Age (yrs)	LVM* g/m ²	Q waves V1-V4	New Glasgow criteria		SV1+RV5/RV6 mV	RI mV	RaVL mV	SV1/SV2 mV	RV5/RV6 mV
JB	M	57	253	V1-V3	+		5.7	1.0	1.1	3.3	3.0
KK	F	66	171	V1-V3	+		4.9	1.8	1.5	2.4	2.7
EB	F	52	134	V1-V2	-		1.8	0.9	1.2	0.5	2.0
AB	M	67	220	V1-V2	+		3.4	1.9	1.6	1.5	2.2
SB	F	52	274	V1-V2	+		3.0	1.0	1.2	1.4	2.2
LT	F	72	141	V1-V2	+		3.8	0.8	1.2	3.8	1.8
JM	M	38	155	V1-V2	-		3.5	0.9	0.6	2.5	2.6
JF	F	39	171	V1-V2	+		4.2	1.7	1.2	1.9	2.1
HS	M	72	197	V1-V2	+		4.3	1.3	1.0	1.7	2.7
GW	M	57	246	V1-V2	+		6.6	1.2	0.8	2.2	4.2
IS	F	38	144	V1-V2	-		1.7	0.65	0.4	0.5	1.4
AN	F	54	305	V1-V2	+		4.2	1.6	1.2	2.3	1.8

* Indexed LVM calculated on Penn Convention

Table 8.18 Voltage criteria (precordial and limb leads) and the new Glasgow criteria in patients with anteroseptal Q waves.

ECG-LVH criteria	Anteroseptal MI without echo LVH	Anteroseptal MI with echo LVH	Echo LVH without anterior MI
Score \geq 4 with new Glasgow criteria	0/13	10/20 (50%)	9/12 (75%)
SV1 + RV5/RV6 \geq 3.5 mV	2/13 (15%)	6/20 (33%)	8/12 (66%)
R I > 1.3 mV	1/13 (7%)	5/20 (25%)	4/12 (33%)
R aVL > 1.1 mV	1/13 (7%)	5/20 (25%)	7/12 (58%)

voltage criteria $R I > 1.3 \text{ mV}$ and $R aVL > 1.1 \text{ mV}$ were positive in 5/20 (25%) patients with anteroseptal MI and echo-LVH compared with 4/12 (33%) for $R I > 1.3 \text{ mV}$ and $R aVL > 1.1 \text{ mV}$ in the patients with echo LVH without MI.

The specificity and the sensitivity of the ECG-LVH criteria were determined within the three groups of the patients who had Q-waves in anteroseptal leads. The Glasgow criteria, i.e. the new scoring system were found to be 100% specific for the diagnosis of LVH in this population of patients with Q-waves in anteroseptal leads with a sensitivity of 59%. The specificity of $SV1 + RV5/RV6 > 3.5 \text{ mV}$ was 85% with a sensitivity of 44% (table 8.19). However, the limb lead voltage criteria ($R I \geq 1.3 \text{ mV}$ and $R aVL \geq 1.1 \text{ mV}$) were more specific (92%) for the diagnosis of ECG-LVH than the Sokolow-Lyon precordial voltage criteria but with lower sensitivities of 28% for $R I \geq 1.3 \text{ mV}$ and 38% for $R aVL \geq 1.1 \text{ mV}$.

8.19.3 Diagnosis of ECG-LVH in the presence of antero-septal infarction

In the group with anteroseptal MI with normal indexed left ventricular masses, there were 2/13 (15%) patients who had false positive ECG-LVH on the Sokolow-Lyon precordial voltage criteria and other 2/13 (15%) patients with false positive voltage criteria in the limb leads. Furthermore, when the individual voltages ($R I$, $R aVL$, $SV1/SV2$, $RV/RV6$) were compared with the age and sex dependent upper limits of normal (table 8.1), it was

found that no patient in this group had R wave amplitudes in the limb leads above normal while 4/13 (30%) and 1/13 (7%) had SV1/SV2 and RV5/RV6 greater than the age and sex matched upper limits of normal respectively.

These results indicate that, (i) amplitudes of R waves in the limb leads are not affected by anteroseptal MI, (ii) anteroseptal MI causes potentiation of individual precordial voltages i.e. SV1/SV2 in 4/13 and RV5/RV6 in 1/13 patients respectively, and (iii) anteroseptal MI potentiates the amplitude of the QS waves in leads V1-V2 four times more commonly than the amplitude of R in V5 or V6. In this study, it is clear that the specificity and sensitivity of the ECG-LVH criteria analysed in a population of patients with QS deflections in some of leads V1-V4 are better than in the other population of patients from which myocardial infarction was excluded (tables 8.14 and 8.19).

Patients with anteroseptal MI without echo LVH have been shown in this study to exhibit potentiation of the precordial voltages leading to false positive diagnoses which theoretically implies that Sokolow-Lyon precordial voltage criteria would be less specific for ECG-LVH in the presence of anteroseptal MI than otherwise. On the contrary this criterion was found to be 82% specific with a sensitivity of 43% in patients with Q waves in the anteroseptal leads without echo LVH (table 8.19) but 64.4% specific and 58% sensitive in the population of patients without Q-wave anteroseptal MI as shown in table

Table 8.19 Specificity and sensitivity of ECG-LVH
 criteria in a population of patients with
 antero-septal Q waves.

ECG-LVH criteria	Specificity	Sensitivity

Glasgow criteria		
(new score)	100%	59%
SV1 + RV5/RV6 \geq 3.5 mV	85%	44%
R I > 1.3 mV	92%	28%
R aVL > 1.1 mV	92%	38%

3.6. The age range of the patients without anteroseptal MI was from 19 to 81 years and included many patients under 30 years while in the second population of patients with anteroseptal MIs, the patients' ages ranged from 32-81 years. However it is well known that the voltages are higher in the younger population < 30 years than in those > 30 years (Macfarlane and Lawrie 1989), and this may explain the lower specificity of $SV_1 + RV_5/RV_6 \geq 3.5$ mV in the population studied and did not include anteroseptal MIs.

The specificity of the Glasgow criteria for LVH (old scoring system) was 77.7% (LVH = score ≥ 4 points) in the patients without anteroseptal Q waves and increased to 100% (new score) in those with Q wave anteroseptal MIs while the sensitivity remained at 59%. This indicates that the new Glasgow point scoring system for the ECG diagnosis of LVH performed well even in a population including anteroseptal MIs.

Murphy et al (1984) reported that the Romhilt-Estes point scoring system and the abnormal P terminal force performed well for the diagnosis of ECG-LVH but they also found that the sensitivity of ECG criteria increased in the presence of MI at the expense of reduced specificity. However, the specificity of the new Glasgow point scoring system is 100% in the presence of anteroseptal MI and is 59% sensitive. These results indicate that the new Glasgow point scoring system performed well in a test group of patients who had anteroseptal MI.

9. CONCLUSIONS

The major aim of this thesis was to re-evaluate the electrocardiographic (ECG) diagnostic criteria for coronary artery disease (CAD) and left ventricular hypertrophy (LVH), on the basis of modern investigative approaches.

In the preceding chapters, computerized 12 lead ECGs have been compared with clinical, echocardiographic (echo), radionuclide and contrast angiographic data. All the ECGs studied were from patients with disorders affecting the left ventricle (LV). The results have been discussed, from which the following conclusions can be drawn:

9.1 SELVESTER'S QRS POINT SCORING SYSTEM PROVIDES A QUALITATIVE ASSESSMENT OF LV FUNCTION IN THE EARLY POST-INFARCT PERIOD

An evaluation of Selvester's 54 criteria/32 point scoring system for the calculation of ejection fractions (EF) from the scalar ECG in patients with myocardial infarction (MI), was presented. This scoring system was of limited value for the accurate prediction of EFs both in acute and old MI (3rd day and \geq 3 months post-infarct respectively). However, in the acute MI group, it was useful for evaluating LV function qualitatively i.e. it separated patients with good LV function from those with LV dysfunction. This is the first study which has assessed this new QRS scoring system for the evaluation

of LV function in patients with MI.

9.2 ECG LEFT ATRIAL OVERLOAD CAN PREDICT SEVERE LV DYSFUNCTION

ECG left atrial overload defined as an abnormal P terminal force in lead V1 (PTFV1) was shown to detect severe LV dysfunction in patients with acute MI (3rd day post-infarct). This is the first study which has shown that a $PTFV1 \geq 8.0$ mV.msec was associated with Technetium-99 EF $\leq 25\%$. The latter abnormality was found in 8/38 (21%) of patients with acute MI and thus has implications for using the point scoring system for the diagnosis of ECG-LVH by decreasing specificity.

9.3 A NEW ECHOCARDIOGRAPHIC CLASSIFICATION FOR LVH/ ENLARGEMENT

Left ventricular masses calculated from M-mode echoes, and indexed to body surface area (LVMI), were utilized as the gold standard for the ECG-LVH evaluation. The Framingham echo classification for LVH/enlargement was tested in 202 consecutive cardiac patients. The latter approach resulted in a significant overlap between concentric and eccentric types of LVH when they were assessed with respect to their echo LV volumes indexed to body surface area (LVVI). Thus a new classification was proposed based on LVMI and LVVI. The new classification did not result in an overlap of concentric and eccentric LVH, while it also detected a small but significant group of patients with decompensated LV i.e. a normal LVMI but with dilated LV at the expense of thin walls. The new

echo classification was used as a gold standard for the ECG differentiation of concentric and eccentric LVH.

9.4 LIMITED VALUE OF SCALAR ECG IN DIFFERENTIATION OF LVH TYPES IN INDIVIDUAL PATIENTS

One of the main purposes of the ECG-ECHO correlative studies in this thesis was to find the ECG parameters that might differentiate between concentric and eccentric LVH. The overall results of these studies in 84 patients with LVH documented by echo, revealed that it is practically impossible to differentiate them using the ECG because:

- (i) the ST-T changes of LV strain described earlier were found to be equally common in both types of LVH;
- (ii) despite variations in the sensitivity of the ECG voltage criteria in concentric and eccentric LVH, none was specific for one or other type of LVH;
- (iii) the multiple logistic regression equations (MLRE) developed to predict the LVVI from the ECG, had unacceptably low specificities and sensitivities for eccentric LVH.

9.5 VARIABLE EFFECTS OF THE LV GEOMETRY ON THE ECG VOLTAGE CRITERIA

It was found that the limb lead voltage criteria ($R I$ and/or $R aVL \geq 2.0$ mV) were 4.5 times more frequently encountered in concentric than in eccentric LVH. On the other hand, in eccentric LVH, precordial voltage criteria ($SV1/SV2$ and $RV5/RV6 \geq 3.0$ mV) were more frequently positive than in concentric LVH. The latter finding can

be explained either by the Brody effect or the influence of the distance of the LV from the chest wall in patients with LVH with LV dilatation (eccentric LVH). However both types of LVH showed exactly similar sensitivities for the Cornell voltage criterion which may indicate that it is an ideal criterion for studies involving LVH regression.

9.6 ANTEROSEPTAL Q WAVE MI DOES NOT POTENTIATE LIMB LEAD VOLTAGES

The effects of Q waves due to antero-septal MI on the limb and precordial voltages were studied with reference to the age and sex dependent upper limits of voltages used in our laboratory. QS deflections of antero-septal MIs diagnosed in our coronary care unit, did not increase the R voltages in leads I and aVL, but they augmented those of the precordial leads, namely SV1/SV2 and RV5/RV6. This led to significant changes being made in the point scoring system for ECG-LVH diagnosis. These included deduction of one point if the precordial voltages were positive in the presence of antero-septal Q wave MI, but such a deduction is not made if the voltage criteria are only positive in the limb leads.

9.7 VALUE OF LV STRAIN FOR ECG-LVH DIAGNOSIS

All the individual ECG voltage criteria included in the point scoring system were compared to ECG LV strain with respect to their relative specificities and sensitivities. LV strain had a higher sensitivity than all the individual voltage criteria at high levels of

specificity. This study was carried out in a population of 129 cardiac patients without myocardial infarction. These findings contributed to a significant change in the scoring system, i.e. the score of well defined LV strain was raised, while the basic score for the voltage criteria was reduced to 2 points only.

However in another population of 24 patients with LVH documented by echo and who had normal coronary arteries as determined by angiography, variable types of ST-T changes in the anterolateral leads were observed. Typical LV strain was found in 71% of these patients. However, atypical ST-T changes in the anterolateral leads were observed in the remaining 29% of these patients. It was thought that these atypical ST-T changes might be in part responsible for reducing the sensitivity of the old Glasgow point scoring system in the diagnosis of LVH. However, it is difficult to differentiate such atypical ST-T changes of LVH with normal coronary arteries from those due to coronary artery disease alone. Therefore it was thought that their inclusion as a criterion in the point scoring system for the diagnosis of LVH, should be supported by another strengthening criterion to indicate LVH. Thus, a new criterion was introduced in which such atypical ST-T changes in the anterolateral leads score 2 points if they are associated with high voltage in the same leads.

9.8 ROLE OF COMPUTERIZED MEASUREMENTS OF 12 LEAD ECG FOR EVALUATION OF ECG-LVH CRITERIA

The ECG-ECHO correlative study for LVH included 129 cardiac patients without myocardial infarction. In order to study all the existing ECG-LVH criteria and the MLREs for the diagnosis of LVH, all the ECG parameters of these patients in addition to their clinical data (age and sex), and echo LVMI needed to be analysed. This in effect means that in excess of 7000 ECG measurements were used. Without computer aided techniques, there would certainly have been faulty measurements and problems in transferring them to a data base.

Computer aid was also sought for the statistical studies including correlations of ECG parameters with LVMI and, for calculation of specificities and sensitivities of all ECG criteria analysed by the Biomedical Computer Program. Needless to say, the assessment and development of the regression equations from these numerous data, would have been impossible without computer assistance. The MLREs introduced by Casale and colleagues did not perform well in our cardiac population. The newly developed MLRE for detecting LVH in our cardiac population, performed better than any other criteria, including the new Glasgow scoring criteria, with regard to both the sensitivity and specificity. However, the fact that similar equations were not reproducible in our study led to the conclusion that such MLREs perform well only in the populations from which

they are developed, and that their performance drops considerably in a test population.

9.9 THE NEW GLASGOW POINT SCORING SYSTEM FOR ECG-LVH

DIAGNOSIS

The first objective assessment of the old Glasgow point scoring system for ECG-LVH diagnosis was presented. Although the specificity of the old system for diagnosis of definite LVH (≥ 6 points) was 91.1% with a sensitivity of 47%, the specificity for possible LVH (≥ 4 points) was unacceptably low at 77.7%. On the basis of the findings mentioned earlier and in the preceding chapters, a new Glasgow point scoring system has been proposed and evaluated in a test series.

With the new Glasgow criteria, possible LVH achieved a higher specificity (86%) and sensitivity (64%). This represented the only situation in the studies included in this thesis in which there was a significant improvement in both the sensitivity and specificity simultaneously. Another evaluation of this new system by the Association Index, showed marked improvement of the Glasgow criteria, as the index rose from 37.6 on the old system to 50.0 for the new system (score ≥ 4 points) - and even to 63 on the CSE test population.

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APPENDIX 1 OLD GLASGOW POINT SCORING SYSTEM FOR DIAGNOSIS OF ECG LVH

LVH CRITERIA

22-jun-88

These criteria are in the form of a score awarded for each test, which will be totalled up to give ESCORE.

If WPW or LBBB or IVCDLB has been detected, omit this section.

1. Amplitude

score is maximum of scores for individual parts.

Each part scores 3 points if true,

+ part a: 1 extra point for every 0.3 mV over the limit,

parts c,d,f: 1 extra point for every 0.5mV over the limit (age 17+ only)

a. The largest R in I or aVL ge an age and sex dependent limit

c. S in V1 or V2 ge an age and sex dependent limit

d. R in V5 or V6 ge an age and sex dependent limit

e. The Lewis Index $[(R + S_I) - (R_{III} + S_{III})] > \text{an age and}$

sex dependent limit. (age 17 and over only)

f. The Sokolov Lyon Index $[S_{V1} + R_{V5}] > \text{an age and sex}$

dependent limit. (age 17 and over only)

Table of sex and age dependent limits for part 1.

AGE	1-2	3-5	6-8	9-11	12-16	
sex	m+f	m+f	m+f	m+f	m	f
I	1.3mV	1.3mV	1.3mV	1.3mV	1.5mV	1.5mV
aVL	0.9mV	0.9mV	0.9mV	0.9mV	1.1mV	0.9mV
V1						
V2	3.0mV	3.25mV	3.5mV	3.5mV	4.0mV	3.5mV
V5						
V6	3.25mV	3.75mV	3.75mV	4.0mV	4.0mV	3.5mV

AGE	17-29		30-39		40-49		ge 50	
sex	m	f	m	f	m	f	m	f
I	1.5mV	1.5mV	1.6mV	1.4mV	1.6mV	1.4mV	1.6mV	1.4mV
aVL	1.1mV	0.9mV	1.2mV	1.0mV	1.3mV	1.2mV	1.3mV	1.2mV
V1								
V2	4.0mV	3.5mV	3.5mV	3.0mV	2.5mV	2.5mV	2.5mV	2.0mV
V5								
V6	4.0mV	2.5mV	3.0mV	2.2mV	2.5mV	2.2mV	2.5mV	2.2mV
Lewis Index	2.5	2.0	2.5	1.8	2.0	1.8	2.0	1.8
S-L Index	5.0	4.25	4.75	4.25	4.5	3.75	4.5	3.75

2 (1 point with digitalis, 3 points without digitalis)
In any of I, V5 or V6:

STj ≤ -0.02 mV, and ST slope $< +5$ degrees (STSLOP ≤ 0), and
T- ≤ -0.2 mV, with T morphology = -1, and
There are no Q waves (see Myocardial Infarction section).

3 (3 points)

P wave flag is set, and
Terminal amplitude of P in V1 < -0.11 mV, and
Terminal duration of P in V1 ≥ 0.04 secs

If no. 3 is not true, and no. 2 did not score 3,
score 1 if atrial fibrillation is present.

4 (2 points)

Inferior infarction not detected, and
Overall QRS axis is between -30 and -120 degrees

5 (1 point)

- a. QRS duration in V5 or V6 ≥ 0.10 secs, and
- b. none of RBBB, RBBB+LAFB, RBBB+LPFB, MASC RBBB, or
IVCD of RBBB type are present

6 (1 point)

Intrinsicoid deflection in V5 or V6 ≥ 60 ms, and
There are no Q waves (see M.I. section) in V5 or V6.

LEFT VENTRICULAR HYPERTROPHY

LVH0=30

ESCORE ≥ 6

PROBABLE LEFT VENTRICULAR HYPERTROPHY

LVH0=20

ESCORE = 5

POSSIBLE LEFT VENTRICULAR HYPERTROPHY

LVH0=10

ESCORE = 4 and there are ST or T wave changes
in the lateral leads

..... WITH SECONDARY ST-T CHANGES

LVSTRN

Add to LVH0=30,20 or 10

If in I, V5 or V6,

ST slope < -10 degrees, and ST < -0.05 mV, and

T- < -0.2 mV,

or similarly in aVL, but with R $> |S|$

..... WITH ST-T CHANGES LIKELY TO
BE DUE IN PART TO MYOCARDIAL ISCHAEMIA

Add to LVH0=30,20 or 10 if the above addition is not true, and
ANTSTT is not true, and

T- < -0.2 mV in aVF with no inferior infarction, or

T- < -0.2 mV in I or V5 or V6 with no lateral infarction,

PROBABLE EXTREME LIMIT OF NORMAL FOR AGE
AND SEX IN VIEW OF CLINICAL FINDINGS

LVH0=21

a. ESCORE ≤ 5 , and

b. any of EST1A, EST1C or EST1D above is true, and

- c. there is no BVH score, and
- d. the patient is under 35 years, and
- e. there are no ST-T change results (STTCH0=-1), and
- f. there are no ST-T reasons for LVH set, and
- if g. Clinical classification is Normal (10),

CONSIDER AS UPPER LIMIT OF NORMAL IF NO
OTHER RELATED CLINICAL ABNORMALITY FOUND

LVH0=22

As above, but instead of g.,

- if h. Clinical classification is not Normal, Hypertension,
Congenital Heart Disease, Rheumatic Heart Disease, or
Cardiomyopathy (10,13,15,17,49),

APPENDIX 2

NEW GLASGOW POINT SCORING SYSTEM FOR

DIAGNOSIS OF ECG-LVH

Feb 6 15:46 1990 ulvh.txt Page 1

LVH CRITERIA

09-nov-89

These criteria are in the form of a score awarded for each test, which will be totalled up to give ESCORE.

If WPW or LBBB or IVCDLB has been detected, omit this section.

1. Amplitude

Points are awarded for high voltage in various leads and the score is the maximum of scores for individual parts (a-f).

Each part scores 2 points if true. However, points can be added or subtracted according to the following rules:

ADD

part a: 1 extra point for every 0.3 mV over the limit,
parts c,d,f: 1 extra point for every 0.5mV over the limit (age 17+ only)

SUBTRACT

parts a - f: 1 less point if Q waves or low R waves in anterior leads
(asm1,2,7,8,12,amipro)

Criteria:

- a. The largest R in I or aVL ge an age and sex dependent limit
c. S in V1 or V2 ge an age and sex dependent limit
d. R in V5 or V6 ge an age and sex dependent limit
e. The Lewis Index $[(R + S_I) - (R_{III} + S_{III})] > \text{an age and sex dependent limit. (age 17 and over only)}$
f. The Sokolow-Lyon Index $[S_{V1} + R_{V5}] > \text{an age and sex dependent limit. (age 17 and over only)}$

Table of sex and age dependent limits for part 1.

AGE	1-2	3-5	6-8	9-11	12-16	
sex	m+f	m+f	m+f	m+f	m	f
I	1.3mV	1.3mV	1.3mV	1.3mV	1.5mV	1.5mV
aVL	0.9mV	0.9mV	0.9mV	0.9mV	1.1mV	0.9mV
V1						
V2	3.0mV	3.25mV	3.5mV	3.5mV	4.0mV	3.5mV
V5						
V6	3.25mV	3.75mV	3.75mV	4.0mV	4.0mV	3.5mV

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AGE	17-29		30-39		40-49		ge 50	
sex	m	f	m	f	m	f	m	f
I	1.5mV	1.5mV	1.6mV	1.4mV	1.6mV	1.4mV	1.6mV	1.4mV
aVL	1.1mV	0.9mV	1.2mV	1.0mV	1.3mV	1.2mV	1.3mV	1.2mV
V1								
V2	4.0mV	3.5mV	3.5mV	3.0mV	2.5mV	2.5mV	2.5mV	2.0mV
V5								
V6	4.0mV	2.5mV	3.0mV	2.2mV	2.5mV	2.2mV	2.5mV	2.2mV
Lewis Index	2.5	2.0	2.5	1.8	2.0	1.8	2.0	1.8
S-L Index	5.0	4.25	4.75	4.25	4.5	3.75	4.5	3.75

2 ST-T changes

a) In any of I, aVL, V5 or V6:

- i) ST_T ≤ -0.020 mV, and ST slope ≤ -5 degrees ;
or ST_T ≤ -0.050 mV, and ST slope ≤ 0 degrees
- ii) |ST_T - T_T| > 0.1mV
- iii) T_T ≤ -0.2 mV, and T morphology < 0 with T₊ < 0.150mV
- iv) R > 1.0mV or R' > 1.0mV
- v) There are no Q waves in the lateral leads
- vi) QRS duration < 120 msec

Score 4 points if: i - vi are true

Score 2 points if: i, ii, iii, v, vi are true

b) If 2a is not true consider the following:

- i) TLAT ≤ -2 or STDPLA ≤ -2
- ii) 1a or 1e is true
- iii) 1c or 1d or 1f and not anterior infarction
- iv) 1c or 1d or 1f and anterior infarction
- v) QRS duration < 120 msec

If i,v and (ii or iii) score 2 points

If i,v and iv score 1 point

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For 2a and 2b

Deduct 2 points if ST-T changes and there is inferior myocardial infarction with T- aVF $< -0.05\text{mV}$

N.B. No account is taken of digitalis in this scoring system

3 P terminal force (2 points)

P wave flag is set, and
Terminal amplitude of P in V1 $< -0.11\text{mV}$, and
Terminal duration of P in V1 ≥ 0.04 secs

Fibrillation/flutter

If above is not true,
score 1 if atrial fibrillation or flutter is present.

4 Left Axis Deviation (2 points)

Inferior infarction not detected, and
Overall QRS axis is between -30 and -120 degrees

5 QRS duration (1 point)

- a. QRS duration in V5 or V6 ≥ 0.10 secs, and
- b. none of RBBB, RBBB+LAFB, RBBB+LPFB, MASC RBBB, or IVCD of RBBB type are present

6 Intrinsicoid deflection (1 point)

Intrinsicoid deflection in V5 or V6 $\geq 60\text{ms}$, and
There are no Q waves (see M.I. section) in V5 or V6.

LEFT VENTRICULAR HYPERTROPHY

LVH0=30

EScore ≥ 6

PROBABLE LEFT VENTRICULAR HYPERTROPHY

LVH0=20

EScore = 5

POSSIBLE LEFT VENTRICULAR HYPERTROPHY

LVH0=10

EScore = 4 and there are ST or T wave changes
in the lateral leads

..... WITH SECONDARY ST-T CHANGES

LVSTRN

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Add to LVH0=30,20 or 10 if LV Strain (i.e. 2 above) is true

..... WITH ST-T CHANGES LIKELY TO
BE DUE IN PART TO MYOCARDIAL ISCHAEMIA

Add to LVH0=30,20 or 10 if the above addition is not true, and
ANTSTT is not true, and
T- < -0.2 mV in aVF with no inferior infarction, or
T- < -0.2 mV in I or V5 or V6 with no lateral infarction,

..... WITH ST-T CHANGES PROBABLY DUE IN PART
TO MYOCARDIAL ISCHAEMIA/DIGITALIS EFFECT

Add to LVH0=30,20, or 10 if:
i) the above is true
or LV strain is present
and ii) digitalis is being administered.

CONSIDER LEFT VENTRICULAR HYPERTROPHY
SUGGESTED BY VOLTAGE CRITERIA ONLY

LVH0=11

- a. ESCORE ge 4, and
b. all of EST2 - EST6 are false
c. there are no ST-T changes in the lateral leads

PROBABLE EXTREME LIMIT OF NORMAL FOR AGE
AND SEX IN VIEW OF CLINICAL FINDINGS

LVH0=21

- a. ESCORE le 5, and
b. any of EST1A, EST1C or EST1D above is true, and
c. there is no BVH score, and
d. the patient is under 35 years, and
e. there are no ST-T change results (STTCH0=-1), and
f. there are no ST-T reasons for LVH set, and
if g. Clinical classification is Normal (10),

CONSIDER AS UPPER LIMIT OF NORMAL IF NO
OTHER RELATED CLINICAL ABNORMALITY FOUND

LVH0=22

As above, but instead of g.,
if h. Clinical classification is not Normal, Hypertension,
Congenital Heart Disease, Rheumatic Heart Disease, or
Cardiomyopathy (10,13,15,17,49),